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DIETETIC TREATMENT OF CONVULSIONS AND ALLIED CONDITIONS OCCURRING IN INFANTS

WITH SPECIAL REFERENCE TO THE RÔLE PLAYED BY INORGANIC SALTS *

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For many years the subject of spasmophilia or infantile tetany has been of great interest to the pediatricians. The modern idea is that spasmophilia is a condition rather than a disease; that in the organism there is the latent possibility of the manifestation of active symptoms of this condition, and that this manifestation may be produced by various factors in the surroundings of the child, but especially by a food which is injurious to a given child at a given time. We see, therefore, that two factors underlie the production of the clinical manifestations of spasmophilia or tetany. The first of these is the diathesis or predispostion; the second, the exciting cause. With the first of these this paper will have nothing to do.

ETIOLOGY

As yet no one has attempted to determine how this spasmophilic predisposition is acquired, nor how limited. It is a generally accepted fact that the most powerful of the exciting causes of the clinical manifestations of spasmophilia is an unsuitable food.

In 1905 Finkelstein noted that the irritating portion of the diet seemed to be in the whey of cow's milk. This finding has been confirmed by me as described in a previous paper,[1] and some examples forming the foundation of my reason for believing in this will be given later. Having been convinced that a food consisting of a carbohydrate with the curds of milk, the whey of which had been thrown away, was relatively non-irritating in these cases, I determined to find out, if possible, what

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1. Arch. Pediat., 1912, xxix, 24.

element of the whey it was which produced the irritating symptoms. It is a general belief, and one which has been most specifically formulated by Reiss,[2] that the calcium and magnesium salts act antagonistically to the sodium and potassium salts, the former tending to allay nervous irritability, the latter to increase it. In other words, the formula $\frac{Ca}{Na}$ or, rather, $\frac{Ca + Mg,}{Na + K}$ would more accurately represent the proposition. When there is a relative increase of Ca and Mg over the Na and K, one would expect, if we follow the reasonings on this subject, to have a decreased nervous irritability, while the opposite proposition would also be true.

The means by which we can most accurately estimate the nervous irritability in infants and animals is by the electrical reaction. It has been definitely determined that in infants suffering with spasmophilia KOC is less than 5 Ma., while AOC is less than ACC. Speaking more specifically, it requires less than 5 milliamperes of current to produce the cathode opening contraction, while the anode opening contraction is produced by less usually than is the anode closing contraction.

Taking the foregoing hypothesis, it was determined to approach the subject of spasmophilia from a somewhat different standpoint from that which has generally been taken in the past. It was determined to endeavor to produce increased electrical irritability by the use of the sodium salts. Before doing this, however, a study of the metabolism of the inorganic salts in animals before and after thyroidectomy (in which latter condition is shown markedly the reaction to the electric current) may be of interest.

EFFECT OF THYROIDECTOMY ON SALT METABOLISM

The following experiments on dogs were undertaken with the idea of determining whether any disturbance in the metabolism of inorganic salts occurred during the period of increased electrical irritability following thyroidectomy. The experiments were carried out in the following way:

TECHNIC

Young dogs were chosen, 1 or 2 months old, preferably females. These dogs were kept in cages such as are ordinarily provided for animal metabolic experiments. The animals were observed for a period previous to operation and a period after operation. In all cases the first period was three days. A two, three or four-day period was observed after operation. The food, which consisted, in all cases, of milk, was preserved by the addition of 1 to 10,000 formaldehyd solution, and at no time showed any gross signs of decomposition. The milk was measured in a graduate and the dog, except in one instance, which will be noted later, was

2. Ztschr. f. Kinderh., 1911, iii, 1.

given as much as it would take. There existed previous to the beginning of the experiment a twenty-four-hour hunger period. The urine and feces were collected each day and immediately put into combustion flasks containing concentrated sulphuric acid. The excreta for the three days were not examined separately, but the feces and urine were each put into the flasks and the total excretion of each for the three days estimated. No attempt was made to estimate the weight of the feces. The urine was estimated in cubic centimeters. The urine and feces were then heated without the addition of nitric acid or other material and the organic matter destroyed. For the estimation of the nitrogen in the milk the ordinary Kjeldahl process was used, while for the other constituents the Neumann method of destroying the organic matter was employed. In all cases the nitrogen was estimated by the Kjeldahl method, the phosphorus by the molybdate method. The calcium was precipitated as calcium oxalate and weighed as calcium sulphate. The magnesium was estimated from the filtrate from the calcium oxalate solution by being precipitated as magnesium pyrophosphate and weighed. For the sodium and potassium a method devised by Koch was used. The sulphuric acid was driven off in a platinum dish, the residue taken up with water and the solution

Dog I

Before Operation

	Food	Urine	Feces	Retention Amt	Retention Percent
N	4.370	2.555	.403	1.412	32.3%
P	1.258	.394	.110	.754	59.94%
Ca	.929	.022	.101	.806	86.76%
Mg	.137	.063	.020	.054	39.42%
Na	.665	.529	.088	.048	7.22%
K	.269	.228	0	.041	15.24%

After Operation

	Food	Urine	Feces	Retention Amt	Retention Percent
N	4.002	2.442	.275	1.185	29.61%
P	.751	.236	.056	.459	61.12%
Ca	.845	.001	.061	.773	91.47%
Mg	.094	.017	.021	.056	59.58%
Na	.554	.179	.049	.326	58.89%
K	.196	.118	0	.078	39.79%

Table 1.—Findings in the metabolism experiment before and after operation on Dog 1.

filtered, the filtrate being evaporated and the residue weighed. From this filtrate the potassium was estimated by the cobalti-nitrite method of Bowser and the sodium content determined by difference.

EXPERIMENTS

Of the twelve dogs on which metabolism experiments were started, it was possible to complete them in only four instances.

Dog 1.—Two months old. Three days before operation weighed 1630 gm. Took three days previous to operation 801 c.c. of milk; passed 502 c.c. of urine. The KOC three days before operation equalled 3.5 Ma. On the afternoon of the third day the thyroid tissue was removed, which was very small in quantity. This dog at no time developed true tetany. On the day after the operation KOC equalled 1.8 Ma. On the second day after operation KOC equalled 1.4 Ma. On the fourth day after the operation KOC equalled .2 Ma. During the four day

period after operation this dog drank 675 c.c. of milk, and passed 422 c.c. of urine.[3] This dog was killed with ether at 10:30 a. m. on the fourth day. A search for thyroid and parathyroid tissue was made and none was found. The weight of the dog at the time of death was 1590 gm.

The only evidence we have of a tendency to tetany in this dog lies in the increased electrical irritability. This dog was in the first series of those examined, and only the cathode opening contraction was taken, the method for doing which will be described later. It will be seen, however, that there was a distinctly increased electrical irritability in this instance; so much so that it renders it more than likely that the parathyroid tissue was removed, although the dog showed no active signs of tetany.

If we examine the table showing the metabolism of the various salts, we find that the nitrogen retention before and after operation, as well as the phosphorus, was practically the same. There was a very slight increase of calcium retention after operation and a distinct increase of magnesium. Neither of these is so marked, however, as are the figures which show the retention of sodium and potassium after operation. If any conclusions are to be drawn from these figures, one might say that in this instance the increased electrical irritability was accompanied by retention in the system of those salts which are commonly regarded as favoring increased nervous irritability; that is, the sodium and potassium, while there was no increase in the retention of those salts which are commonly regarded as allaying the electrical irritability, namely, the calcium and magnesium.

Dog 2.—Male dog, 2 months old. This dog was given daily 200 c.c. of milk, both before and after operation. In the three days previous to operation the dog passed 634 c.c. of urine. In the four days after operation, it took 800 c.c. of milk, and passed 151 c.c. of urine. Unfortunately, the weight was not taken until after operation, but on the third to the fifth day after operation the weight decreased from 2750 gm. to 2505 gm. The electrical reactions previous to operation were also unsatisfactory in this dog, so that we can draw no definite conclusions as to increase of electrical irritability. However, on the fourth day this dog developed distinct tetany and was found dead on the morning of the sixth day. The brain and spinal cord of this dog were removed and their composition appears under Dog 5 in Table 3 on the composition of the brains.

In this dog there was no question of the development of tetany. It had distinctly developed previous to the conclusion of the metabolic experiment. For some reason or other there was in this case previous to operation an extremely low nitrogen retention for which I am unable

3. The ammonia content of the urine was estimated in this dog for three days before and three days after operation. In the three days before operation the ammonia output was as follows: First day, 141.6 mg. NH_3 (following a starvation period of twenty-four hours). Second day, 38.4 mg. NH_3. Third day, 41.2 mg. NH_3. After operation: First day, 31.8 mg. NH_3. Second day, 54.9 mg. NH_3. Third day: 58.2 mg. NH_3. The estimates were made by the Folin method.

to account. The phosphorus retention, too, was reduced. As in the previous case, the calcium retention was practically the same before and after operation, and in this dog there was, as in the previous one—though not so marked—an increased retention of magnesium after operation. While before operation in this dog there was a negative sodium and potassium balance, after operation the sodium retention was very marked, while the potassium still remained negative. The result in this dog would seem to give one the same impression as that in the case of Dog 1.

BRAIN FINDINGS IN A SERIES OF DOGS

Following the metabolic experiments on the first two dogs, it was thought advisable to check up the brain findings of other investigators. As previously stated, Dog V in Table 3 is the brain of Dog 2 of the previous series of metabolic experiments.

Dog II

Before Operation

	Food	Urine	Feces	Retention Amt	Retention Per cent
N	3.233	2.747	.228	.258	7.98%
P	.611	.364	.021	.226	36.99%
Ca	.632	036	.032	.564	89.25%
Mg	.141	.027	.003	.111	78.72%
Na	.893	1.091	.051	-.249	—
K	.278	.690	0	-.412	—

After Operation

	Food	Urine	Feces	Retention Amt	Retention Per cent
N	4.311	1.124	.259	2.928	67.92%
P	.814	.141	.056	.617	75.80%
Ca	.842	.020	.115	.707	84.09%
Mg	.188	.007	.011	.170	90.20%
Na	1.191	.267	.047	.877	73.64%
K	.370	.514	0	-.144	—

Table 2.—Findings in the metabolism experiment before and after operation on Dog 2.

The other four brains of Table 3 were taken from dogs of the same litter, which were approximately 1 month of age. Dog I of this table was used as a control and was killed on the eighth day. No operation was done on this dog and in the eight days it gained in weight from 2,650 gm. to 3,110 gm. The brain and spinal cord weighed 62.1 gm.

Dog II of Table 3 weighed at the beginning of the observation 2,570 gm. On the third day KCC equaled 3.5 Ma., and KOC equaled 3.5 Ma. One the fifth day KCC equaled 1.5 Ma.; KOC equaled 7.5 Ma. At 10 o'clock on the morning of the fith day the thyroid was removed. The day following operation KCC equaled 1.5 Ma.; KOC equaled 2.0 Ma., at

8:30 a. m. At 9:10 a. m., 5 c.c. of a 10 per cent. calcium lactate solution was injected with the result that immediately afterwards KCC equaled 1.2 Ma.; KOC equaled 4.0 Ma. After the injection of the calcium the dog was depressed and vomited a great deal. The next day at 8:30 a. m. the electrical irritability was: KCC, 1.6 Ma.; KOC, 4.5 Ma. Immediately after taking the electrical irritability 10 c.c. of a 5 per cent. calcium lactate solution was injected intravenously. At 10:30 KCC was 4.6 Ma., and KOC, 8.3 Ma. At 9 a. m., on the next day, the KCC was 1.5 Ma.; KOC, 2.6 Ma. Five c.c. of a 10 per cent. calcium lactate solution was immediately given, after which the dog seemed to be much more lively. At 10 a. m. the KCC equaled 2.6 Ma.; KOC equaled 3.5 Ma. At 8 a. m. on the following morning the dog showed very marked tetany, with KCC 1.1 Ma.; KOC, 3.0 Ma. The dog was killed and the brain and cord removed. Weight, 59 gm.

Brain	Total Weight	Ca	Mg	K	Na	
Dog I	62.1gms	.0045	.0009	.0209	.2590	Control
Dog II	59 gms	.0033	.0044!	.0174	.2254	
Dog III	61 gms	.0031	.0002	.0231	.1652	
Dog IV	67 gms	.0024	.0002	.0253	.2775	
Dog V	65 gms	.0033	.0002	.0255	.3048	

Table 3.—Results of examination for various inorganic salts in the brains and spinal cords of five dogs, Dog 1 being a control.

Dog III (Table 3), was a female weighing 2835 gm. Second day of observation: KCC equaled 2 Ma.; KOC equaled 2 Ma. Fifth day: KCC equaled 3.3 Ma.; KOC equaled 1.4 Ma. Sixth day: KCC equaled 1 Ma.; KOC equaled 2.6 Ma. Seventh day: KCC equaled 3 Ma.; KOC equaled 3 Ma. Operation at 10 o'clock.

First day after operation, KCC equaled 3.1 Ma.; KOC, 6.9 Ma. On the second day following, convulsions. KCC equaled 2.4 Ma.; KOC equaled 3.1 Ma. On the third day, during tetany, KCC equaled 2 Ma.; KOC equaled 2 Ma. The dog died on the evening of the third day after operation. Weight of brain and cord, 61 gm.

Dog IV (Table 3), was a male weighing 2865 gm. On the fifth day of observation KCC equaled 2 Ma.; KOC, 6.5 Ma. On the seventh day, KCC equaled 6.5 Ma.; KOC, 6.5 Ma. Operation at 10 o'clock on the seventh day.

After operation: First day, KCC, 7.5 Ma.; KOC, 5.5 Ma.

The dog died in tetany on the morning of the second day before electrical irritability could be tested. Weight of brain and cord, 67 gm.

Table 3 gives the results of examination for various inorganic salts in the brains and spinal cords. It will be noted that in regard to the calcium the findings agree perfectly with those of other investigators.

There is shown a distinct decrease in the calcium content in all dogs, the first dog being the control. As to the magnesium, the decrease is most marked, except in Dog II of this table, which dog received repeated injections of calcium lactate. I have no way of explaining this finding. It has been suggested that this may have been due to an error in estimation. This might be an acceptable explanation, except that the amount of calcium as shown in this dog agrees very well with that found in Dogs III and V.

There is very little to be said in regard to the potassium and sodium, both remaining varyingly constant throughout.

We see, then, that even in a dog such as Dog V, Table 3, where the calcium retention was not reduced, the calcium content of the brain was markedly lowered.

If we determine the quotient in the formula $\frac{Ca}{Na}$ we find that in the control dog it was about 58, while in the four other dogs it was respectively 68, 53, 115 and 92. We see, therefore, that Dog III, Table 3, does not conform to this formula, although the calcium is distinctly reduced[4].

The metabolism experiments were now renewed.

Dogs 3 and 4 of the metabolism experiments were of the same litter and 1 month of age.

Dog 3, male, gives the following detailed history:

Dog 3.—Aged 1 month; sex, male; color markings, black and white.

TABLE 4.—DATA CONCERNING EXPERIMENTAL DOG 3

Day		Weight, Gm.	Milk, c.c.	Temp. Cent.	——Electrical Irritability—— KCC.	KOC.	ACC.	AOC.	Urine c.c.
	Feb. 12—								
1	10 a. m.	2180	50	38.3	10+	10+	10+	10+	95
			100	....	....	....	....	....	...
2	.....	2170	90	38	10+	10+	10+	10+	45
			100	....	....	....	....	....	...
3	.....	2300	90	39	10+	10+	10+	10+	112
			75	....	....	....	....	....	...
4	10 a. m.	2050	85	38	10+	10+	10+	10+	...
			590	....	....	....	....	....	252
	Operation about 3 p. m., Feb. 15.								
	Began collections at 8 a. m., Feb. 16.								
1	.....	1970	75	40	4	4	2	4.5	30
			60	....	....	....	....	....	...
2	.....	1890	90	....	....	....	....	....	27
			225	....	....	....	....	....	57

Found at 8 a. m., February 18, just breathing. Two full days of collections. Some tetany on morning of February 17. At autopsy, blauder contracted and contained no urine.

4. These results do not differ from those of Aschenheim. Monatschr. f. Kinderh,. 1910, ix, 366.

Table 5 shows result of the metabolism experiments on Dog 3. In this dog the most marked metabolic changes recorded are to be found, first, in the decreased nitrogen retention after operation, which is accompanied by a decreased phosphorus retention. There is also a distinctly lessened calcium and magnesium retention after operation, together with a lowering in the retention of the sodium and potassium. It is to be noted, however, that the lowering of the retention of calcium and magnesium is greater than is that of the sodium and potassium.

Dog III

Before Operation

	Food	Urine	Feces	Retention Amt	Retention Percent
N	3.966	1.985	.354	1.627	41.02%
P	.738	.277	.038	.423	73.00%
Ca	.765	.046	.049	.670	87.52%
Mg	.086	.037	.018	.031	35.70%
Na	.895	.404	.104	.387	43.34%
K	.875	.471	0	.404	46.32%

After Operation

	Food	Urine	Feces	Retention Amt	Retention Percent
N	1.293	1.019	.275	0	0
P	.241	.118	.034	.089	36.81%
Ca	.249	.025	.052	.172	68.80%
Mg	.028	.022	.010	−.004	—
Na	.292	.113	.091	.088	29.84%
K	.285	.238	0	.047	16.51%

Table 5.—Findings in the metabolism experiment before and after operation on Dog 3. (This dog and Dog 4 (Table 7) belong to the first series of (metabolism) experiments.)

Dog 4.—Aged 1 month; sex, female; color markings, yellow and white.

Table 6.—Data Concerning Experimental Dog 4

Day	Weight, Gm.	Milk, c.c.	Temp. Cent.	Electrical Irritability KCC.	KOC.	ACC.	AOC.	Urine c.c.
Feb. 12—								
1	1950	30	38	8	8	10	10	18
		75	....	....	....	....	....	...
2	1970	75	38	10+	10+	10+	10+	47
		75	....	....	....	....	....	...
3	1880	75	38	10+	10+	10+	10+	19
		75	....	....	....	....	....	...
4	1850	75	38	10+	10+	10+	10+	...
		480	....	....	....	....	....	84
Operation, 3 p. m., Feb. 15.			Tetany.					
1	1800	75	38	3.5	3.5	3.5	3.5	24
		40	....	....	....	....	....	...
2	1700	90	35.2	1.5	1.5	1.5	1.5	0
		205	....	....	....	....	....	24

February 18, 8 a. m.: Dog in extreme tetany. No urine in last twenty-four hours. Killed by bleeding. Bladder absolutely empty.

Table 7 shows the metabolic changes in Dog 4. In this dog, as in Dog 3, there is a distinct decrease in nitrogen retention. The decrease in phosphorus retention, however, is very much less marked and can be disregarded. The decrease in calcium retention is slight, and if we take the figure that it is necessary to observe (a 20 per cent. difference) in metabolic experiments, in order that the figures may be of any value, we may disregard here, too, the calcium retention. The magnesium retention before operation was about 20 per cent. greater than after operation. The sodium was practically the same before and after, while the potassium showed a slightly increased retention before operation over that after operation.

Dog IV

Before Operation

	Food	Urine	Feces	Retention Amt	Retention Per cent
N	2.765	.679	.194	1.892	64.82%
P	.513	.125	.051	.337	65.71%
Ca	.532	.027	.041	464	87.14%
Mg	.060	.025	.009	.026	41.94%
Na	.495	.082	.086	.327	66.01%
K	.609	.157	0	.452	74.28%

After Operation

	Food	Urine	Feces	Retention Amt	Retention Per cent
N	1.179	.569	.169	441	37.40%
P	.219	.055	.032	.132	59.96%
Ca	.227	.014	.042	.171	75.40%
Mg	.026	.010	.010	.006	22.34%
Na	.266	.009	.076	.181	68.01%
K	.260	.130	0	.130	50.00%

Table 7.—Findings in the metabolism experiment before and after operation on Dog 4.

If we now sum up the findings in these four dogs, we see that perhaps the first three fall in very well with the opinion of Reiss, that the increased electrical irritability is accompanied by disturbance of the salt equilibrium, in that there is either a deficiency of the calcium and magnesium, or an excess of sodium and potassium, or both. But this theory is decidedly not upheld in any way by the findings in the last dog. It will be noted that this dog died in extreme tetany. There can be no doubt of the increased electrical irritability, and yet from the metabolic experiments herein noted no conclusions can be drawn which would involve the salt equilibrium as being the active factor in producing the clinical picture.

EXPERIMENTS ON ELECTRICAL IRRITABILITY AFTER INJECTIONS OF SODIUM AND CALCIUM SALTS

Following the above two sets of experiments, it was deemed advisable to test the electrical irritability after injections of sodium and calcium salts, both before and after removal of the parathyroid. It is a well-known fact that life can be prolonged in a parathyroidectomized dog by the injection of sufficient quantities of calcium solution. It has never been possible, however, so far as I know, to prevent the final fatal result by such methods. The amount of calcium necessary to produce such results is far in excess of any quantity which could be excreted in a given length of time by the organism. The action of calcium in this instance, therefore, in no way proves that the symptoms resulting from parathyroidectomy are due in any measure to an increased excretion of calcium. In fact, magnesium salts act in general the same as do the calcium salts, and if we may distinguish between a physiological and a pharmacological action of the inorganic materials in the system, we would certainly characterize the calcium and magnesium action in this instance as pharmacological. It was not thought likely, and the experiments proved that my position was right, that there would be such a difference in the time required for death to intervene after thyroidectomy, in order to distinguish between the actions of the various salts.

Some rather comprehensive experiments on electrical irritability were, therefore, undertaken. I will describe the technic in detail because of the irregular results obtained.

TECHNIC

The electrical irritability, except where noted to the contrary, was always taken between 8:30 and 9:30 in the morning, after the dogs had been fed their morning meal. The food given the dogs consisted of scraps containing some meat, but mostly carbohydrates. The dog's head and fore-paws were held by one assistant, the hind legs by another. The electrodes, of which one was a metal plate ½ by 1½ inches, the surface covered with several layers of gauze, the other an iron point covered with gauze, were first well saturated with normal salt solution and then applied to the abdomen, the larger electrodes being placed near the umbilicus, the smaller in the inguinal fold, and at a measured distance from the large electrode. This distance was determined in each instance by the use of a small piece of wood which had been measured for the purpose. In each instance, then, the electrodes were the same distance from each other, were applied on a portion of the skin which was free, or practically free, from hair, and the slightest twitching of the muscles in the region was regarded as a positive result. The other operator read the milliampere meter and made and broke the circuit.

The first six dogs of this last set of experiments were of the same litter and were 1 month of age. The last dog was somewhat larger and was 6 weeks of age. Dog 1 (Table 8) was used as a control, and weighed 5900 gm. The temperature during the week of observation varied from 39 to 40.5 C. At the end of the seventh day the dog's weight was 5720 gm. Both thyroids, weighing 1.33 gm. were removed. The dog was observed three days after operation. On the third day the weight had fallen to 5250 gm. The temperature after operation was somewhat lower than previous to operation, the last two days not going above 38 C. The dog was anesthetized with ether and bled to death. The accompanying table (Table 8) shows the results of the electrical irritability.

It will be seen from this table that these results were very irregular; that the greatest irritability occurred on the third day before operation and the second day after, and that for three days previous to operation the electrical irritability was much lower than at any time after operation.

Dog I

	Before Operation			
Day	KCC	KOC	ACC	AOC
1	1.5	.75	3.0	4.5
2	1.2	2.0	1.2	7.0
3	1.0	1.0	1.5	2.4
4	—	—	—	—
5	3.6	4.7	5.5	9.2
6	2.8	2.8	4.0	5.5
7	2.8	8.2	4.9	6.2
	After Operation			
1 AM	0.8	1.4	0.6	0.6
1 PM	0.6	2.5	1.1	1.1
2	1.0	1.0	0.6	1.3
3	1.6	4.2	2.4	2.4

Table 8.—Tests for electrical irritability, expressed in milliampères, before and after operation on Dog 1. (Dogs 1 to 7 of Tables 8 to 14 constitute a different group of dogs from those reported in previous tables.)

Dog II

	Before Operation			
Day	KCC	KOC	ACC	AOC
1	.75	5.0	.75	—
2	2.0	1.0	1.0	8.2
3	0.4	0.5	1.8	2.7
4	2.0	3.5	2.8	2.8
5	2.0	5.5	3.5	3.5
6	1.4	5.0	2.6	4.0
7	1.0	1.5	1.5	2.4
	After Operation			
1 AM	1.3	1.3	1.2	1.2
1 PM	1.5	1.5	1.0	2.0

Table 9.—Tests of electrical irritability, expressed in milliampères, before and after operation on Dog 2.

Dog 2 (Table 9) weighed on the first day of observation 4820 gm. On the seventh day, 4500 gm. The temperature varied between 38.2 C. and 41 C. Each day this dog received morning and evening an intraperitoneal injection of 20 c.c. of a normal sodium acetate solution. In the afternoon of the seventh day both thyroids, weighing .75 gm., were removed. In the afternoon of the second day

following the operation the dog was in violent tetany, and was immediately killed. The temperature the first week was 39 C. to 41 C., and the weight on the afternoon of the day following operation was 4350 gm.

It will be seen from the table that the electrical irritability was extremely irregular before operation and that the findings following operation show no marked increase of electrical irritability over those before.

Dog 3 (Table 10) weighed at the beginning of the experiment 4440 gm. On the seventh day its weight was 4350 gm. The temperature varied between 38.2 C. and 40 C. This dog received morning and evening 20 c.c. of a normal solution of sodium lactate intraperitoneally. The dog was operated on during the afternoon of the seventh day. Both thyroids were removed, weighing 1.58 gm. This dog lived seven days, and was found dead the morning of the eighth. For the first four days the tetany was rather marked. After that the dog lapsed into a semi-comatose state, in which condition it died. After operation the temperature varied from 38.5 to 39.5 C. On the first and second days after operation the dog received 20 c.c. each of a normal sodium lactate solution. In this dog a comparison of the electrical irritability before and after operation shows a rather marked increase following the removal of the thyroids.

Dog III

	Before Operation			
Day	KCC	KOC	ACC	AOC
1	4.0	7.5	5.0	7.5
2	2.5	4.0	2.5	——
3	0.5	2.5	2.2	2.9
4	2.2	2.2	2.7	4.0
5	2.5	3.4	6.5	6.5
6	3.1	9.0	5.4	5.4
7	3.7	6.0	6.0	7.0
	After Operation			
7 AM	1.4	6.9	2.7	1.5
7 PM	1.0	1.0	1.7	1.7
2	1.6	2.5	1.6	1.4
3	0.4	1.0	0.6	0.6
4	0.9	1.2	0.5	1.2

Table 10.—Tests of electrical irritability, expressed in milliampères, before and after operation on Dog 3.

Dog 4 (Table 11) at the beginning of observation weighed 4900 gm. On the afternoon of the seventh day the weight was 4450 gm. The temperature varied between 39 and 40 C., and reached 40 C. only the afternoon of the seventh day. This dog received 20 c.c. of a normal sodium chlorid solution intraperitoneally morning and evening. Both thyroids were removed, the weight of which was 12.23 gm. The vessels were found very brittle and hard to tie. The dog died on the third night after operation. The temperature after operation was 39 C., and the weight on the afternoon of the second day was 4350 gm. The dog received 20 c.c. of a normal sodium chlorid solution intraperitoneally on each day after operation. It had distinct tetany on the day following operation. Although the first day after operation the electrical irritability was not greatly increased, the second day showed a distinct increase of electrical irritability.

Dog 5 (Table 12) weighed at the beginning of observation 5200 gm. On the afternoon of the seventh day, 4900 gm. Temperature varied between 38 and 40.5 C. On the afternoon of the seventh day both thyroids weighing 1.73 gm., were removed. This dog was anesthetized and bled to death on the evening of the third

Dog IV

Day	KCC	KOC	ACC	AOC
	Before Operation			
1	4.0	6.0	4.0	6.0
2	3.7	3.7	3.4	9.5
3	6.5	5.5	7.5	7.5
4	5.0	5.0	4.2	4.2
5	9.8	9.8	10.0	12.5
6	2.4	4.6	3.5	——
7	5.7	5.7	5.5	5.7
	After Operation			
1	4.5	5.7	11.0	11.0
2	1.2	1.2	1.6	1.6

Table 11.—Tests of electrical irritability, expressed in milliampères, before and after operation on Dog 4.

Dog V

Day	KCC	KOC	ACC	AOC
	Before Operation			
1	2.0	3.5	3.5	9.5
2	2.4	2.4	2.5	10.0
3	1.1	1.5	1.8	2.4
4	2.0	3.0	1.8	2.5
5	6.5	6.5	7.5	7.5
6	2.5	8.0	4.5	5.0
7	2.5	5.7	6.0	6.0
	After Operation			
1 AM	1.5	4.5	2.0	4.5
1 PM	2.7	4.5	2.0	13.5
2	1.1	1.4	0.5	1.0
3	0.6	1.1	0.5	0.5

Table 12.—Tests of electrical irritability, expressed in milliampères, before and after operation on Dog 5.

day. It received previous to operation one injection on the first day of 18 c.c. of a normal sodium lactate solution, and 2 c.c. of a normal calcium lactate solution. After that it received 20 c.c. of a normal sodium lactate, and 2 c.c. of a normal calcium lactate solution, morning and evening, on each day before opera-

tion. After operation, on the morning and afternoon of the first day and the morning of the second day it received 18 c.c. of a normal sodium lactate solution and 2 c.c. of a normal calcium lactate solution. This dog developed distinct tetany on the afternoon of the first day, and was killed while in tetany.

A glance at the table will show that there was a marked increase of electrical irritability in this dog, especially on the third day after operation.

Dog 6 (Table 13) weighed on the first day of observation 4740 gm.; on the seventh day, 4500 gm. Temperature varied between 39 and 40 C. Operation on the afternoon of the seventh day. The thyroid glands, weighing 12.58 gm., were removed. The vessels were found to be very brittle. Before operation this dog received morning and evening 18 c.c. of a normal sodium acetate solution, and 2 c.c. of a normal calcium acetate solution. On the day following operation the dog had slight tetany. It received an injection of normal sodium acetate solution, 18 c.c., and calcium acetate, normal solution, 2 c.c. On the afternoon of the second day the dog was having a distinct tetany and received again the same injection. The dog died during the night and weighed, the following morning, after death, 3850 gm.

Dog VI

	Before Operation			
Day	KCC	KOC	ACC	AOC
1	3.5	4.5	3.5	4.5
2	2.9	2.9	3.0	10.0
3	1.5	1.5	1.5	1.5
4	3.2	7.2	7.2	7.2
5	2.5	4.3	3.0	6.7
6	3.3	4.7	3.0	11.5
7	2.0	3.5	3.0	4.0
	After Operation			
1	6.0	9.0	5.4	13.0
2	12.5	12.5	To high to register	

Table 13.—Tests of electrical irritability, expressed in milliampères, before and after operation on Dog 6.

Peculiar in this dog is the very low electrical irritability following operation. This was much lower both days after operation than on any day previous to operation. It is hard to explain this condition, but it may have been due to the fact that the severe tetany had so exhausted the muscles that they refused to react to the ordinary currents.

Dog 7 (Table 14) weighed on the first day of observation 6200 gm.; on the seventh day, 5750 gm. The temperature varied between 39 and 40.2 C. This dog received morning and evening 24 c.c. of a normal sodium chlorid solution, and for the first four days 2.5 c.c. of a normal calcium chlorid solution. On the fifth day the dog was given 4 c.c. of normal calcium chlorid solution; on the sixth day, 2.5 c.c., and on the seventh day, 3.5 c.c. On the afternoon of the seventh day the thyroid gland, weighing 1.11 gm., was removed. This dog showed no tetany on the first day after operation. On the second day the tetany was so violent that it was impossible to take an electrical reaction. The temperature stood at 42 C. and the stools were bloody. The weight was 5300 gm. This dog was anesthetized and bled to death. This dog, in the few electrical reactions which it was possible to take after the operation, showed a distinct tendency to an increased electrical irritability.

Peculiar to all seven of these dogs was the very irregular reaction to the electric current before operation. However, it can be said that with the exception of Dog 6 there was a distinct increased electrical irritability following operation in all instances. As previously stated, the explanation for the failure of this increased electrical irritability to occur in Dog 6 cannot be given satisfactorily. It is seen here, too, that the injection of sodium solutions before and after operation could in no instance be said to have definitely increased the electrical irritability, nor to have lessened the interval between the operation and death. The calcium solutions evidently had very little effect.

Dog VII

Day	KCC	KOC	ACC	AOC
	Before Operation			
1	4.5	4.5	3.5	7.6
2	5.5	5.5	5.4	15.0
3	3.5	3.5	3.0	3.0
4	2.8	5.0	5.0	5.0
5	3.4	4.0	5.8	8.0
6	7.8	12.0	7.5	13.0
7	3.0	8.0	4.5	4.5
	After Operation			
1 AM	1.4	3.0	3.8	2.8
1 PM	1.5	1.5	3.0	3.0

Table 14.—Tests of electrical irritability, expressed in milliampères, before and after operation on Dog 7.

From these injection experiments one can only draw negative conclusions. It would seem that the injection of such solutions intraperitoneally, in no way which can be estimated, either increased or decreased the symptoms following removal of the thyroid glands. In all of these animal experiments it was impossible for me to draw any definite conclusions as to why the tendency to tetany in infants is so markedly reduced when the whey is removed from the food. That such is the case I think the following clinical observations will definitely indicate:

CASE REPORTS

CASE 1.—Baby S., four months old, of German parentage, entered Provident Hospital on the afternoon of March 6, 1912. This patient was referred to me by Dr. H. B. Thomas, and had been under his care for one week, suffering with repeated convulsions. The weight on entrance was 11 pounds, 4½ ounces. At the time of entrance the child had a convulsion which lasted about three minutes. The electrical reaction showed a KOC of 2.5 Ma. This child was given a colonic flushing, and one grain of chloral hydrate every four hours rectally, the food consisting of barley water. During the night the child had two more convulsions.

On the morning of the seventh the child was ordered a food consisting of the curds of 30 ounces of milk in 30 ounces of arrowroot water, with 1 grain of saccharin. The electrical irritability on the afternoon of that day showed KOC over 5 Ma. The chloral hydrate was continued during this day. The child had no more convulsions; the chloral hydrate was continued as previously. The electrical irritability on the eighth remained over 5 Ma. The same was true of the ninth. On the tenth the food was changed to 20 ounces of albumin milk, 10 of barley water, and one grain of saccharin, giving a slight increase in the whey content of the food. The chloral hydrate was continued, as previously. The electrical irritability was not taken. On the following day, however, the chloral hydrate being continued as previously, the KOC was 4 Ma., and the child had a slight convulsion in the afternoon. On the twelfth the food was again changed back to the curds of 30 ounces of whole milk in 30 ounces of arrowroot water, and one ounce of dextrimaltose was added. That afternoon the electrical irritability was over 5 Ma. No more convulsions occurred. The chloral hydrate was continued as previously. The next day, the chloral hydrate being continued, and cod liver oil, ½ dram, three times a day, being added, the electrical irritability remained over 5 Ma. On the following day, the fourteenth, the chloral hydrate was discontinued. The electrical irritability still remained over 5 Ma. This was true of the fifteenth. On the sixteenth, 5 ounces of buttermilk was added to the food. The electrical irritability still remained over 5 Ma., and the child went on to an uneventful recovery, the buttermilk being added to the amount of 15 ounces on the seventeenth, and the food being changed to albumin milk the following day. On the day of discharge, the fourteenth day, the child weighed 11 pounds.

It will be seen that in spite of the chloral hydrate there was a distinct tendency to increase of electrical irritability in the early stages of this trouble on the addition of as small an amount of whey as is represented in 20 ounces of albumin milk; in other words, the amount of whey contained in 10 ounces of buttermilk.

CASE 2.—Baby S., 5 months of age, negro, entered Provident Hospital on the evening of March 2, 1912. The child was having convulsions when brought in by the father. The KOC equaled 2.5 Ma. On entrance the child was given 3 grains of chloral hydrate rectally. On the day following the child was given the curds of 18 ounces of milk in 30 ounces of arrowroot water, and one grain of saccharin. The child had no more convulsions, and seemed to be in excellent condition, the temperature remaining normal. On the sixth the food was changed to 20 ounces of albumin milk, 10 of barley water, and ½ ounce of dextrimaltose. On the afternoon of that day the KOC equaled 1.5 Ma. The following day the formula was changed to the curds of 20 ounces of whole milk, 30 ounces of arrowroot water, and 1 grain of saccharin. The electrical irritability on the afternoon of that day was KOC, 3 Ma. On the morning of the following day, KOC equaled over 5 Ma. The child remained in good condition and was taken out by the parents on the morning of the ninth.

Here, too, we see a child coming in with marked convulsions which, after a single injection of chloral hydrate, ceased and showed no tendency to reappear during the period in which the child was given a food without any whey. On the day on which the whey was again added to the food the child showed a very great and rather alarmingly increased electrical irritability, which promptly declined to normal within thirty-six hours after the whey was removed from the food. The objection that may be raised to this observation is that during the period when the

electrical irritability was greatest dextrimaltose had also been added to the food, but, as the previous experiment shows, and as all former evidence has proved, the addition of carbohydrates to the food during the convulsive stages, if given in reasonable quantity, in no way tends to promote an increased electrical irritability.

It seems to me that these two cases are conclusive evidence, after the four clinical observations which I have previously published,[1] that removal of the whey from the food gives a food which does not tend to produce an increased electrical irritability. No one can claim anything but a negative action for such food. In other words, the removal of whey from the food removes those constituents which tend to increase electrical irritability and, therefore, the convulsive tendency.

It was now determined to see if by the addition of the sodium and potassium salts to the curd mixture it would be possible to increase the electrical irritability.

CASE 3.—Baby B., aged 17 months, entered Cook County Hospital on April 3, with a diagnosis of convulsions. The convulsions were first noticed about two months before entrance. At that time they occurred about twice during the week. They had been increasing in frequency ever since until on the day before entrance the child had three convulsions. The appetite was poor and constipation had been quite marked. The general health had always been good previously. The child was a well-nourished colored child. There were marked signs of rickets. The liver and spleen were not palpable. On entrance, the child weighed 16 pounds; temperature was 99 F. The child had no more convulsions after entrance. From the day of entrance it was given barley water until the following afternoon at 2 o'clock, when it was given a diet consisting of curds of 30 ounces of whole milk and 40 ounces of arrowroot water. This diet was continued throughout the experiment. The second day after being placed on this food this child was given 5 grains of sodium chlorid in each of the five feedings; on the following day 5 grains of sodium chlorid and 10 grains of potassium chlorid in each feeding. This was discontinued on the eighth. The electrical reactions are best shown in Table 15.

TABLE 15.—ELECTRICAL REACTIONS IN CASE 3

Date	4/4	4/5	4/6	4/7	4/8	4/9	4/10
KCC	2.0	2.0	3.5	2.5	2.5	1.5	2.0
KOC	2.0	5+	5.0	4.0	4.5	3.5	4.0
ACC	2.5	4.0	3.0	2.5	3.5	4.5	5.0
AOC	2.0	4.0	3.0	2.7	3.0	3.0	3.0
			NaCl	NaCl + K Cl			

CASE 4.—Baby C., aged 5 months, entered Cook County Hospital April 27 at 2 o'clock in the morning. The child had been fed on malted milk since birth. Four days previous to entrance the child began having convulsions; it had several convulsions during the night; had been constipated, and flushings were of no avail. During the examination the child had another convulsion. No other findings of any consequence. The temperature on entrance was 99.8 F. The child was placed on barley water, and the following morning on 16 ounces of whole milk, 9 of barley water, ¾ ounce of malt extract, and ¾ ounce of dextrimaltose. At 6 o'clock in the afternoon the child had a convulsion, which was repeated at 8 o'clock, and at this time no laryngismus nor Chvostek's sign were noted. On the twenty-eighth at 10 a. m. the food was changed to curds of 25 ounces of whole milk; arrowroot water, 25 ounces; malt extract, ¾ ounce, and dextrimaltose, ¾

ounce. The child had convulsions at 8 a. m., 9:35 a. m., and 2 p. m. At 2:30 a grain of chloral hydrate was given rectally. At 6:30 the child had another convulsion lasting a minute. At 7 the child was given another grain of chloral hydrate per rectum. On the twenty-ninth the food was again changed to 16 ounces of whole milk and 14 of barley water, the malt extract and dextrimaltose remaining the same. On the morning of the thirtieth the child had another convulsion, and was put on barley water for twenty-four hours. No convulsions were noted following the barley water. On May 1 the food was changed to the curds of 30 ounces of whole milk, ½ ounce dextrimaltose, and 30 ounces of arrowroot water. On the second it was noted that there was some laryngismus present. On the third the food was changed to the casein fat milk of Heim, which consists of the curds of a quart of whole milk to which a pint of whole milk is added, the whole made up to a quart. The child had no further convulsions and left the hospital on the fifth in good condition. The weight on entrance was 11 pounds, 8¾ ounces, and when dismissed 11 pounds, 5 ounces.

In this case the electrical irritability was taken and is shown in Table 16.

TABLE 16.—ELECTRICAL REACTIONS IN CASE 4

Date	KCC	KOC	ACC	AOC
4/29	2	3	3	2
4/30	5	2	2.5	2
5/1	1	5+	3	1
5/2	4	5+	6	2

On the second, 4 grains of sodium chlorid in solution was added to each bottle. The electrical irritability is shown in the table.

It should be stated that the electrical irritability was taken at 8 p. m. each evening. It will be noted that the addition of sodium chlorid to the food in no way affected the steady tendency to betterment of the electrical irritability.

CASE 5.—Baby B., entered Provident Hospital Sept. 9, 1912, at 11 a. m. On the evening before entrance the child began to have convulsions and had had convulsions every twenty minutes from 11 o'clock on. At the time of entrance the child's temperature was 105.6 F. It continued high during the first twenty-four hours, dropping to between 99 and 100.4 on the next day, and on the third, and on the fourth day becoming normal in the morning and rising to 101 F. in the afternoon. This afternoon temperature continued until the day before the child left the hospital, when the afternoon temperature was only 99 F. On entrance KOC equaled 2.5 Ma. The child was put on barley water for twenty-four hours, at the end of which time KOC was over 5. For the next twenty-four hours the child was put on the curds of 32 ounces of milk and 40 ounces of arrowroot water, with an ounce of dextrimaltose. On the third day the electrical irritability still remained normal. On the morning of the third day 5 grains of sodium chlorid and 10 of potassium chlorid were given in each bottle. This was continued for three days without any effect on the electrical irritability. It was then removed with like result.

In this case one certainly can not draw the conclusion that the addition of sodium and potassium to the food had any effect whatever on the electrical irritability.

These findings do not conform to those of Rosenstern,[5] who obtained an anodic hyperirritability by administering 100 c.c. of 3 per cent. NaCl

5. Jahrb. f. Kinderh., 1910, lxxii, 154.

solution *per os* to spasmophilic infants in every case in which he obtained a rise in temperature. In some cases after the disappearance of the laryngeal spasm, the child being on breast milk, this returned after two or three days, when 6 to 8 grams of sodium chlorid per day were given. The experiments of Rosenstern differ in two main points from those which I have just recorded. First, the anodic hyperirritability was obtained only when a fever temperature resulted from the administration of sodium chlorid solution; and in the second set of cases the amount of sodium chlorid was far in excess of that which I used. It will be noted in my cases that the amount of sodium chlorid and potassium chlorid corresponded very closely to the amounts of these salts which would have been found in the quantity of whey corresponding to the amount of curds used. Considering the results obtained by the use of wheyless mixtures in spasmophilia, it seems to me that this last method of procedure is to be preferred, since it most closely approaches the conditions clinically encountered.

CONCLUSIONS

1. In dogs in which a hyperirritability of the nervous system existed, as shown by the increased electrical irritability, and as produced by removal of the thyroid gland, there is no regular variation in the formula, $\frac{Ca}{Na}$ or $\frac{Ca + Mg}{Na + K}$ before and after operation, as shown by metabolic experiment. While in three dogs thus examined there was apparently some support of the proposition that the quotient of this formula is increased during the period of hyperirritability of the nervous system; in the fourth dog no such reaction could be noticed.

2. In estimating the salt content of the brains of five dogs, the first being a control, there was regularly found a decrease in the calcium content in the thyroidectomized animals. In one instance, however, the formula $\frac{Ca}{Na}$ was less than that of the control animal.

3. No variation in electrical irritability nor severity of the convulsive period could be demonstrated by intraperitoneal injections of normal sodium and calcium salts in the quantity of 40 to 45 c.c. of sodium salts, and 2 to 4 c.c. of calcium salts in twenty-four hours.

4. Even under normal conditions, when carefully estimated, the electrical irritability in dogs varies quite widely.

5. While food containing whey is distinctly irritating to spasmophilic infants, the sodium and potassium salts corresponding in quantity to those contained in the whey do not regularly produce the increased electrical irritability which one would expect were the sodium to be regarded as the irritating substance in the whey.

6. This failure of the sodium and potassium to produce results may be explained either by the fact that the forms in which the sodium and potassium were given, i. e., NaCl and KCl, were not readily absorbed in the gastro-intestinal canal, or that these salts were not in themselves the irritating factors. The latter proposition would seem to be much more probable.

It will be seen, therefore, that these experiments have brought little or no confirmation of the hypothesis that the increased electrical irritability in spasmophilia is the result of a disturbance of calcium and sodium equilibrium.

I wish to acknowledge here my great indebtedness to the late Professor Waldemar Koch for superintending the chemical analyses and for many valuable suggestions, and to Professor S. A. Matthews for supervising the experimental work; also to my interns at the Cook County Hospital for their assistance.

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THE SIGNIFICANCE OF THE PYLORIC REFLEX IN TRUE AND PSEUDOPYLORIC STENOSIS IN INFANTS

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The following case study is presented with the hope that it may illustrate how a regard for the opening pyloric reflexes may serve us in the medical cure of pyloric stenosis and pyloric spasm in young infants.

Methods used in the treatment of the present case are in no way new. It has been known for a long time that lavage and the antacids have been useful and effective in the treatment of these conditions and the explanation for their beneficial effects have been well interpreted. There are, however, a few principles which may be said to be comparatively new which are not generally considered and which make the interpretation of our previous success with these methods still more clear. These are the factors which govern the opening and closing of the pylorus. While we have no definite comparative experiments to prove it, it might well be argued on clinical grounds that in the condition of hyperchlorhydria, accompanying a hypertrophic stenosis of the pylorus or a stenosis from any other cause, in that stage of the disease when spasm can be demonstrated, the irritated tissues respond more markedly to the reflex stimuli. We should at least be able to demonstrate in a stenosis which is not complete, yet of marked degree, whether or not the reflex mechanism is still intact.

We have seen experimentally in the normal infant's stomach[1] how an antacid many be employed to keep the pylorus closed and thus impede the passage of chyme from the stomach by its action in delaying the opening reflex. Under certain conditions a paradoxical reaction may be said to take place; that is, an antacid may be employed to facilitate or expedite the passage of chyme from the stomach. When, for example, an antacid is added in proper amount to a hyperacid stomach, instead of delaying the opening reflex, as is the case in the normal stomach in which achlorhydria is the rule, the opening reflex is brought into play and kept in activity by an early series of short duodenal closings, brought about by the antacid overcoming the excess of acidity or delaying its rapid appearance. Thus, during the first part of digestion one could expect the stomach to empty itself quickly; during the latter part when the acidity has reached its height, more slowly. The question of whether or not we are dealing with a hypersecretion or a simple hyperchlorhydria is at this point of particular importance and should be determined. In the

1. Cowie and Lyon: Am. Jour. Dis. Child., 1911, ii, 252.

former condition the continuous flow of gastric juice leaves in an otherwise empty stomach an acid fluid, which, if neutralized before the meal enters, would, theoretically, facilitate at least a second or a third pyloric opening. With an anacid duodenum a pyloric opening should come as soon as the acidity of the chyme reaches a certain point and that point we have shown, in the infant, is before the appearance of free acid. When free acid appears the duodenal closing is prolonged, depending on the ability of the duodenum to dissipate its acquired acidity. Hence, as stated before, we might expect a lower stomach motility or propulsion force during the latter part of digestion.

In the consideration of the subject of infantile pyloric stenosis I must necessarily confine myself to that group of cases in which hyperchlorhydria plays an important part, for it is with this group alone that I have had experience. A word may be said at this point. It is my belief that a very much larger per cent. of the cases of infantile pyloric stenosis and pseudostenosis is associated with hyperchlorhydria than is usually conceded. I form this belief not from a large experience with the disease in infants, as I have thus far seen but six cases, but from data which I have gathered during a long and somewhat careful experience in the observation of hyperchlorhydria in the adult, and of stomach analyses in infants and children. Such observations disclose that stomach vagaries are alike not uncommon in infants, young children and adults. The same food stimulus while it calls forth an equal amount of juice to saturate it each time it is placed in the stomach, yet the stomach may differ in its response, as indicated by the degree of acidity, within very wide limits from day to day. Although in the adult this fact was early pointed out by Ewald, only comparatively recently has it been taken into consideration in the diagnosis and treatment of stomach states. It has not been taken into account in many of the cases of infantile pyloric stenosis recorded as cases of achlorhydria. In all of the cases I have seen there have been times when no test for free acid could be obtained. I made the statement some years ago, and it may not be out of place to repeat it here, that a single observation of the stomach juice is of no more significance in the diagnosis of stomach disease than a single observation of a thermometer in the diagnosis of a fever. Spasm of the pylorus does occur with achlorhydria in adults and in infants. It has been my privilege to see a number of cases among adults, and in one carefully studied case, which I should like to record, actually to see the spasm occur twice during laparotomy, a pyloroplasty by Professor Darling and myself. Immediately following the tight contraction of the pylorus, which becomes as hard as a piece of India rubber, the peristaltic waves of the stomach brought the organ almost completely out of the abdominal wound. It receded and after a short interval went through a similar but less marked phase.

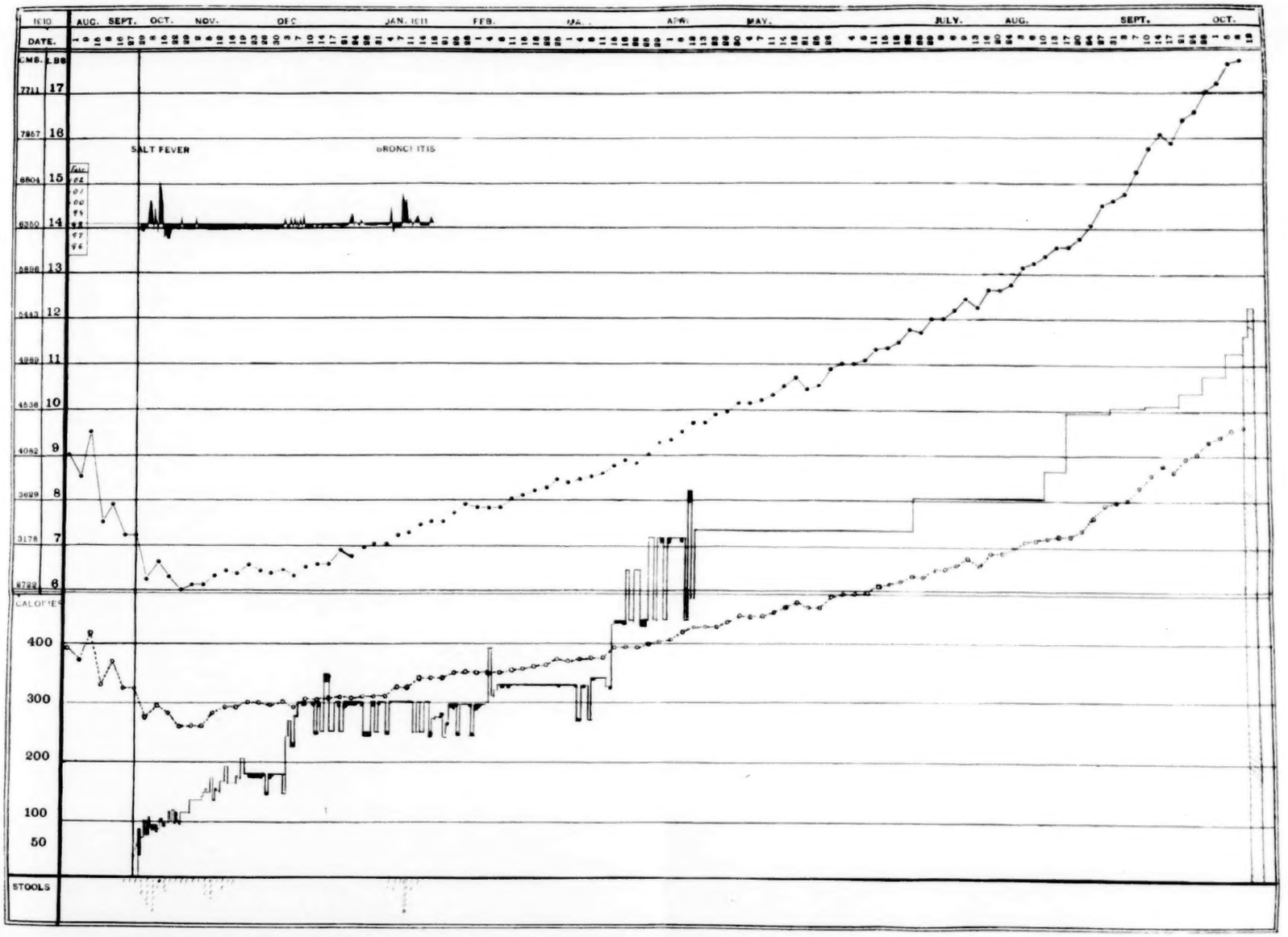

Chart 1.—This chart gives the weights from birth to one year, two months. The dotted line is the energy line (calories required). The food blocks start below the energy line. The dark part indicates the amount of food vomited, the white part the amount retained. The weight begins to rise as the food approaches the energy line.

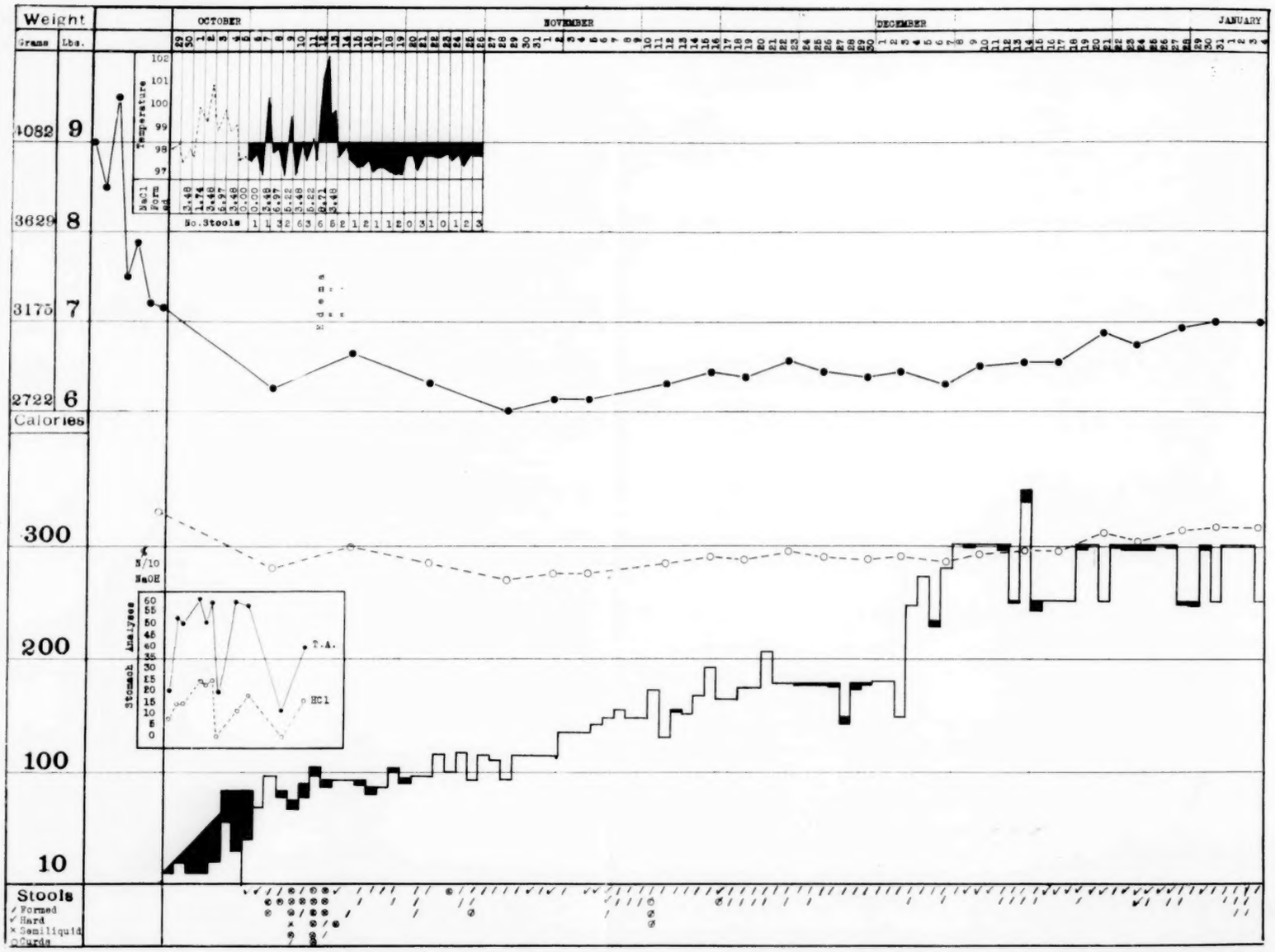

Chart 2.—Detail of first three months of Chart 1. Temperature curve shows a salt fever. Below the curve the number of grams NaCl formed from the $NaHCO_3$ in stomach. Solid line, weight. Dotted line, energy line, indicating caloric requirements. Below, the calories of food given and retained are indicated. The black portion indicates the amount of food vomited, the light portion the amount retained.

CASE REPORT

History.—Sept. 29, 1910. Robert L. Born Aug. 1, 1910; normal birth. Well developed boy weighing nine pounds. Bottle fed from birth. The initial loss of weight was regained in fourteen days. The infant seemed to be developing normally, with the exception of few and hard stools, until August 14, when he first vomited. He had been fed on a formula of milk and milk-sugar obtained from the milk-sugar package. He was now put on condensed milk. The vomiting continued whenever food was given, often in large amounts. The vomitus was frequently "very sour." The vomiting seems to have been allayed for short periods. He retained malted milk feedings at one time for four or five days, and another similar food for one week. Vomiting, however, recurred and wet nursing was tried for ten days; the vomiting, however, persisted. Various other "methods" of feeding were tried but from the fourteenth day of life the infant continued to lose progressively in weight with the report September 29 that it had but two natural bowel movements since birth. September 27 the patient had what was called a "sinking spell." There was marked stupor and general relaxation of the body which lasted a few minutes and was apparently overcome by whiskey. The infant now vomited everything that was put into its stomach. The bowel movements were very infrequent, very small, dry, dark green, with no curds.

Family history negative. Two other children normal.

Examination.—When first seen by me Sept. 29, 1910, the infant was much emaciated, the skin hung in folds over the extremities and its elasticity was practically absent. There was no panniculus. The skeleton was well formed. Fontanels normal. Muscles extremely wasted. No enlarged lymph-nodes. Mucous membranes were somewhat reddened. The lips were dry. The thymus could not be satisfactorily outlined. Thyroid negative. Thorax negative. The general appearance was that of a case of marked "atrophy."

The abdomen was scaphoid in appearance, fuller in the epigastrium than below the navel. On first examination there was a suggestion of peristalsis high in the epigastrium, but not positive. A hard mass about 2.5 cm. in diameter was grasped between the fingers 4 cm. above the navel and a little to the right. It was smooth and felt like an hypertrophied pylorus. There was no pitting on pressure. I failed to find the mass on subsequent examinations. Distinct but small peristaltic waves were observed high in the epigastrium on several occasions but careful watching did not enable me to see the wave pass progressively from left to right. There was distinct separation of the recti abdominalis down to two fingers below the umbilicus.

Stomach Examinations.—(See Table 1.)

Urine Examination.—(See Table 2.)

Feeding Record.—September 29 to October 3 plain whey. October 3 a small amount of cream was added making the formula approximately a 0.4 per cent. fat, 5 per cent. sugar, 1 per cent. protein; to this enough sodium citrate was added to prevent calcium para-casein curds. The strength of the food was gradually increased. November 2 he was getting 1.2 per cent. fat, 5 per cent. sugar, 1.6 per cent. protein. From now on the whey was gradually replaced with water and completely discontinued December 4, at which time he was getting 2 per cent. fat, 5 per cent. sugar and 1.6 per cent. protein. The fat per cent. was increased December 6, 3 per cent. fat, 5 per cent. sugar, 1.6 per cent. protein; January 15, 3 per cent. fat, 5 per cent. sugar, 2 per cent. protein. Citrate of soda was discontinued March 8, 1911. Whole milk was reached Oct. 8, 1911.

Weight Record.—(See Charts 1 and 2.) September 30 the infant weighed 6 pounds 8 ounces.[2] The total loss of weight during observation was 8 ounces. From

2. This record does not appear on the charts. It was lost with the records before referred to. October 8 I find a note saying the baby has lost 4 ounces since last weight.

TABLE 1.—STOMACH ANALYSIS

Observation No.	Date	Amount Fed, c.c.	Interval Since Last Feeding, Hr. Min.	Amount Recovered, c.c.	Excess of Amt. Fed, c.c.	Condition of Stomach Before Feeding	HCl	Total Acid	$NaHCO_3$ with Feeding, gm.	Remarks
	1910									
1	9/29	45	3:00	45	0	.....	8	24	0.0	Sustained duodenal closing reflex.
2	9/30	45	3:00	56	11	.....	20	56	0.0	Sustained duodenal closing reflex.
3	*	45	3:00	53	8	3 hours prev., empty.	20	52	0.0	Sustained duodenal closing reflex.
4	.....	45	3:00	0	0	Empty	..	..	2.5	Released duodenal closing reflex.
5	10/ 1	45	3:00	0	0	.....	..	..	2.5	Released duodenal closing reflex.
6	.....	60	3:00	0	0	.....	..	..	2.5	Released duodenal closing reflex.
7	10/ 2	60	3:00	35	0	.....	20	62	0.0	Sustained duodenal closing reflex.
8	.....	60	3:00	58	0	Vomit. large amt.	18	52	0.0	Sustained duodenal closing reflex.
9	10/ 3	60	3:00	74	14	Four feedgs. since lavage	20	60	0.0	Sustained duodenal closing reflex.
10	.....	60	3:00	30	0	Empty	0	20	2.5	Delayed pyloric opening reflex.
11	10/ 4	60	3:00	0	0	.....	..	..	0.0	.....
12	10/ 5	60	3:00	36	0	.....	12	60	0.0	Sustained duodenal closing reflex.
13	10/ 6	60	3:00	130	70	Vomit. large amt. during night.	18	58	0.0	Sustained duodenal closing reflex.
14	10/ 7	60	3:00	0	0	Previous feed 2.5 gm. carb.	+	—	0.0	.....
15	10/ 8	60	3:00	0	0	.....	..	..	2.5	.....
16	10/ 9	60	3:30	14	0	.....	0	12	0.0	Delayed pyloric opening reflex.
17	10/10	60	3:30	15	0	2.5 gm. carb. previous meal.	..	..	0.0	.....
18	10/11	60	3:30	4	0	.....	22	45	2.5	Released duodenal closing reflex.
19	10/12	60	3:30	0	0	.....	..	..	0.0	.....
20	10/13	60	3:30	0	0	2.5 gm. carb. previous meal.	..	..	0.0	.....
21	10/17	60	3:30	11	0	.....	..	..	0.0	.....
22	10/25	60	3:30	0	0	.....	..	..	0.0	.....
23	10/26	60	3:30	0	0	.....	..	..	0.0	.....
24	11/29	98	3:30	13	0	.....	48	88	0.0	Sustained duodenal closing reflex.
25	11/30	98	3:30	5	0	.....	..	..	0.0	.....
26	12/ 1	98	3:40	0	0	.....	..	..	0.0	.....
27	12/ 2	98	3:40	12	0	.....	40	..	0.0	.....
28	12/ 3	98	3:40	0	0	.....	..	..	0.0	.....
29	12/ 7	120	4:00	2	0	.....	..	..	0.0	.....
30	12/10	120	4:00	3	0	.....	..	..	0.0	.....
31	12/13	120	4:00	0	0	.....	..	..	0.0	.....
	1911									
32	2/27	150	4:00	1	0	.....	++	..	0.0	.....
33	3/23	158	3:00	6	0	.....	10	..	0.0	.....

*Time of Observation 2, 10 a. m.; 3, 1 p. m.; 4, 4:15 p. m.; 5, 11 a. m.; 6, 5 p. m.; 7, 12 m.; 8, 6 p. m.

the end of the first month on there was a steady gain in weight. The ratio increasing in proportion to the proximity of the food line to the energy line. The birth weight was reached March 22 (Fig. 1), seven months, twenty-two days after birth. The child's development is now perfect and he ranks if anything in advance of babies of his age.

DISCUSSION OF CASE

Owing to loss of the nurse's temperature record for the first seven days I am unable to give a true curve of the temperature during that time. The dotted line in the temperature curve, Chart 2, I have improvised. The patient came under treatment with a sub-normal temperature. This continued until the second day of the administration of sodium bicarbonate, when the temperature was slightly above the normal line, and rose to 101 F. the following evening, after which it gradually

TABLE 2.—URINE EXAMINATIONS

Date of Sample	Color, Etc.	Reaction	Specific Gravity	Albumin	Sugar	Acetone	Diacetic Acid	Sediment
1910								
10/ 3	Yellow	Alk. +	1,026	0	0	0	0	Negative.
6	Yellow	Alk. +	.....	0	0	++	—	A few large and small hyaline casts.
14	.....	Alk. +	1,028	0	0	0	0	More similar casts than before. Not large numbers.
17	Pale	Alk.	1,010	0	0	—	—	A few hyaline and epithelial casts.
21	Pale	Acid	1,007	0	0	—	—	An occasional hyaline cast.
24	Pale	Alk.	1,005	0	0	—	—	Negative.
26	Pale	Alk.	.....	0	0	—	—	Negative.
11/ 1	Pale	Alk.	.....	0	0	—	—	Negative.
12/13	Pale	Alk.	1,011	0	0	—	—	A few hyaline and granular casts.
						—	—	
1911								
2/13	Pale	Acid	1,000	0	0	—	—	Negative.

declined to subnormal. Accurate temperature records begin October 6, and are represented by solid black. Subnormal temperature had been frequently noted before I saw the patient. The third and fourth days after beginning the sodium bicarbonate the fever developed. The following day I suspected I was dealing with a salt fever resulting from the sodium chlorid formed from the action of the stomach acid on the sodium salt. The $NaHCO_3$ was discontinued for two and one-half days as a control. The temperature dropped and followed the subnormal type, which will be seen was the characteristic temperature curve of this patient, and which continued to be.

October 7 the sodium bicarbonate was resumed. It was followed by a quite prompt pyrogenic reaction; but although the drug was continued the temperature curve was irregular, at times subnormal, at other times

hypernormal. The dose was increased so that on October 12 as much as 8.71 grams of sodium chlorid would result from the complete action of hydrochloric acid on the sodium bicarbonate given. A marked pyrogenic reaction, 102.2 F., developed along with the formation of this large amount of salt and continued through the following day, during the latter part of which the sodium bicarbonate was discontinued. From now on no carbonate was given and the characteristic subnormal temperature curve was resumed. Synchronous with the temperature rise there was a marked edema of the extremities (water retention), increase in weight and frequent stools, all of which subsided after discontinuing the sodium salt. It will be further observed that during the period of alkali administration free acid appeared in the stomach (Chart 2, and Table 1). There can hardly be any question but that the sodium bicarbonate was completely changed by the acid of the gastric juice. The figures below the temperature curve, Chart 2, show the number of grams of sodium chlorid formed in the stomach. Chart 3 records the total daily amount of alkali given. We have here a marked instance of a salt fever brought about by the administration of a salt, which, when given normal babies in the amount of 1 grain, produces no pyrogenic reaction.[3] The patient was getting full whey as food. It might be argued that the whey salts had something to do with the pyrogenic reaction. However, whey continued to be the bulk of the food until November 2 and was not completely discontinued until December 6. Throughout this entire period the subnormal type of temperature predominated, a febrile reaction occurring only during an attack of bronchitis, as shown on Chart 1.

The Action of the Alkali on the Pyloric Reflex.—In Table 1 in the column devoted to remarks, I have given an interpretation of the conditions found in the stomach at the time of the observation and of the happenings prior to the introduction of the stomach tube. I believe Observations 1, 2, 3, 7, 8, 9, 12 and 13 are reliable illustrations of sustained duodenal closing of the pylorus; Observations 4, 5, 6 and 18, illustrations of released duodenal closing brought about by the alkali, and Observations 11 and 13, illustrations of delayed pyloric opening.

We have shown experimentally how the opening of the pylorus may be delayed in a normal infant by giving it an alkali in its food. A similar result might follow the administration of too much alkali in an infant with hyperchlorhydria. Hence the importance of regulating the dose of acid by means of stomach analyses. We may fail in our treatment of pyloric stenosis and spasm by giving too much or too little antacid. One may unwittingly do damage to the organism by the use of antacids by

3. Cowie, D. M.: Am. Jour. Dis. Child., 1912, iv.

the development of a fever, edema and diarrhea, not suspecting the antacid to be at the bottom of it all.

I think there can be no question that in the present case the rather quick and sure benefit came from the controlled used of the alkali. In six days from the beginning of treatment over two-thirds of the amount given was retained, and on the ninth day the total amount given was retained. See Chart 3.

September 30. Three carefully planned observations followed each other successively (Observation 2). At 10 a. m., three hours after 45 c.c. of whey, 56 c.c. were recovered; the stomach was carefully washed and drained and 45 c.c. of whey given. At 1 p. m. (Observation 3), three hours later, 53 c.c. were recovered. This represented the 45 c.c. of food and 8 c.c. of secretion. These two tests showed that absolutely nothing had passed the pylorus in six hours. The stomach was again drained and (Observation 4) 45 c.c. of whey and 2.5 grams of sodium bicarbonate were given. At 4:15 p. m., three hours later, the stomach was completely empty, the duodenal closing reflex having been released. (All feedings given by gavage.)

From the standpoint of vomiting and emaciation the case was a marked one. For several days before treatment was begun apparently nothing passed the pylorus. Citrate of soda was added to the food as soon as milk was given. Its effect was uniform in making it possible for us to control the size of the curd, and by so doing to recover in most instances the entire stomach contents.

The Urine (Table 2).—As only one urine examination was made prior to the administration of the alkali it cannot positively be said that it or the resulting salt fever was the exciting cause of the casts. However, the only time casts occurred in sufficient numbers to cause concern, was the day following the highest temperatures recorded, October 14. By October 26 the urine was free from casts. A few were found December 13. Albumin tests were always negative. The records in Table 2 show that more careful routine examinations would be of value in such cases.

High Milk Protein Feeding.—Red meat protein is the strongest stimulant for the gastric acid secretion of which we know. Milk protein, on the other hand, stimulates the flow of acid much less markedly and has an enormous binding power for acid. With this fact in mind, it was my desire at the outset to discontinue the alkali as soon as possible and replace it with a high protein mixture. Satisfied, however, with my success and remembering that wisdom is the better part of valor, I cautiously reached 1.6 per cent., November 2, and 2 per cent. January 15, three months after beginning treatment. From now on the gain was marked and the regurgitation less marked. Theoretically, high milk

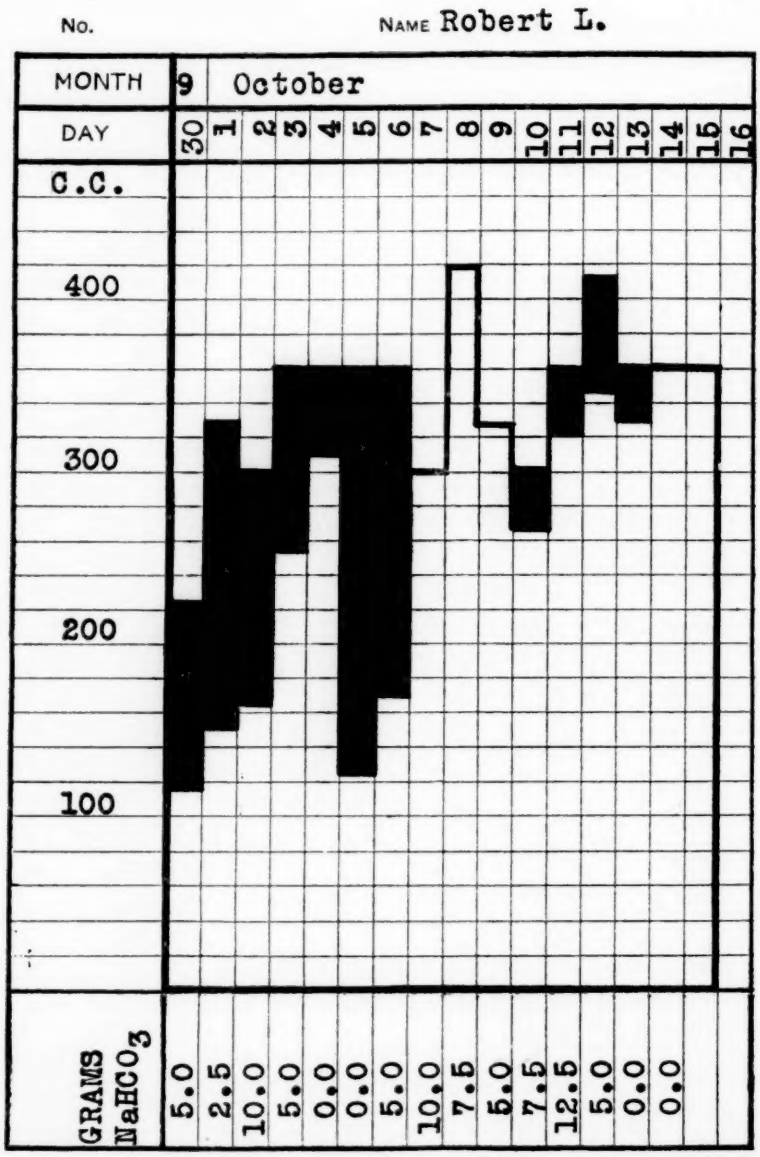

Chart 3.—Showing the amount of food and total daily amount of sodium bicarbonate given during the first sixteen days of treatement. Black indicates the number of c.c. vomited.

protein combined in such a way as to prevent large curd formation in the stomach would be advisable in cases of spasm or stenosis with hyperchlorhydria. The effect of high protein on the kidney epithelium should not be forgotten. It should be remembered that high fat food tends to slow stomach evacuation as the duodenal closing reflex has been shown to be excited by fat in the duodenum.

Stomach Washings.—The object of the study of this case as stated before was to determine how much a regard for the opening and closing reflex could serve us in overcoming pyloric spasm. I think we are all agreed that in these cases associated with hyperchlorhydria, spasm is always present. To this end I endeavored, in so far as was feasible, to exclude stomach washings. Accordingly thorough lavage was resorted to a very few times. The tube nevertheless was passed at frequent intervals to determine the evacuation rate of the stomach. After expressing the contents a bulbful of water was washed through to assure us that all had been recovered. After the first few days this was done once a day or once in two days for a number of weeks and doubtless it had a beneficial effect. Just preceding the present case I treated a severe case by thorough stomach washings before each meal, six times daily. The patient made almost equally as good progress, but the necessary care was almost unendurable for the skilful mother and nurse. By this method we were assured of at least two opening reflexes. The first before the chyme contained free acid, and the second because the acquired duodenal acidity was probably normal and was normally overcome. In this latter case for some time after beginning the treatment we always recovered greater or smaller amounts of food at the end of the four-hour period. I am sure if we bear this reflex action in mind we shall have more uniform success in the care of our cases. Cure ultimately means that we have produced conditions favorable to normal pyloric function.

STATISTICAL COMPARISON OF THE MORTALITY OF BREAST-FED AND BOTTLE-FED INFANTS *

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The great importance of breast-feeding has long been known, but so far the subject has failed to secure its proper share of attention.

Advertisements of infant foods and an abundance of medical literature on scientific feeding of infants have lulled both mothers and physicians into a false sense of security in the practice of artificial feeding. The fearful loss of infant life is so spread out over the entire country that the individual physician does not appreciate his own responsibility, though a conservative estimate ascribes a full third of all infant deaths to unnecessary bottle feeding.

During the siege of Paris, 1870-71, when the milk-supply failed, the Parisian women nursed their children and the infant mortality-rate fell from 330 to 170 per thousand births.[1]

A similar fall in the infant mortality-rate was seen "during the Lancashire cotton famine, when mothers were not at work in the mills."[2]

An analysis of 13,952 children born in Baudeloque's clinic showed an infant mortality of 14 per cent. for the breast-fed, 31 per cent. for those who were bottle-fed by their own mothers and 50 per cent. for those who were bottle-fed by strangers.[3]

And so throughout the civilized world, wherever a large percentage of mothers suckle their children, the infant mortality-rate is low. Norway and Sweden are examples with low rates, respectively, of 74 and 85 per 1,000 births.[4]

But the objection has been made that some mothers are unable to nurse their children.

L. Emmett Holt[5] estimated that not over 25 per cent. of the well-to-do and cultured of New York City who had earnestly and intelligently attempted to nurse had succeeded in doing so for as long as three months.

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*Read at the International Congress on Hygiene and Demography, Washington, D. C., September, 1912.

1. Brehmer: Wchnschr. f. Säuglingsfürsorge, Leipzig, 1907, p. 209.
2. Devine: Hospital, London, 1906-7, xli, 137.
3. Jaschke: Monatschr. f. Geburtsh. u. Gynäk. 1908, p. 172.
4. Victorian Year Book, 1909-10, p. 133.
5. Quoted in St. Louis Med. Rev. 1910, N. S. IV, 324.

At the other extreme are the figures of Mme. Dluski,[6] who in the maternity department of Professor Pinard in Paris, found 99 per cent. able to nurse their children; and between these extremes are found many estimates, as 66 per cent. for France, 90 per cent. for parts of New York City,[7] 96 per cent. for Munich, and so on.[3]

In view of these widely different estimates, in view of the fact that mothers in Greenland, Turkey and Japan almost always nurse their children, and in view of the fact that wherever the propaganda has gone forth that mothers ought to nurse their children greater numbers have been able to do so, is it not reasonable to believe that almost all mothers if properly cared for and encouraged will be able to nurse their young? Such is the belief of Dr. Jacobi,[8] who recently said: "One hundred per cent. of our women can be made to nurse, even the 'flower and fashion' of the land." If such an opinion is too optimistic, certain it is that with proper care and encouragement many more mothers will be able to nurse their children than do so to-day.

For in some places the percentage of mothers who actually suckle their children is extremely low; in Berlin 55 per cent. of mothers nursed in 1885, and but 31.4 per cent. in 1900; while in Leipzig 45 per cent. of mothers either do not nurse at all or less than eight days, and only 27 per cent. nurse their children six months. On the other hand, the possibility of increasing these percentages is well shown by the figures from the Heidelberg Clinic, where in three years the percentage of mothers nursing their children increased from 64 per cent. to 86 per cent.[3]

Investigation of breast and bottle feeding in Boston and its bearing on the infant mortality of the city was recently undertaken by the Boston Health Department.[9]

To find the percentage of babies that were breast-fed about 900 letters were sent to mothers who had births recorded within the past year. Care was taken to select proportionate numbers of babies of various age periods, namely, 2 weeks to 1 month, 1 to 3 months, 3 to 6 months, 6 to 9 months and 9 to 12 months, and to select proportionate numbers of babies from the various wards and from the various mother nativities, the number of births in 1910 in the various wards of various mother nativities being taken as the basis for these proportions. Aside from obtaining the proportionate numbers of babies in the way just indicated, care was taken not to select these babies in any way but to take them at random from the birth returns furnished by the registry department. The circular sent to each mother was as follows:

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6. Thése de Paris, 1894; quoted by Griffith, Bull. Am. Acad. Med., 1910, xi, 151.
7. Hermann: Pediatrics, New York, 1909, xxi, 425.
8. Jacobi: Jour. Am. Med. Assn., June 8, 1912, 1744.
9. Davis: Boston Med. and Surg. Jour., clxvi, 242.

Dear Madam: To assist the Board of Health in its campaign to save the babies of Boston, will you please answer the following questions regarding your child and return this slip at once in the enclosed envelope?

Is this child breast fed? (Answer yes or no.)
How long was it breast fed?
Is this child bottle fed? (Answer yes or no.)
How long has it been bottle fed?
If bottle fed, what food has been used?

Replies were received promptly from over half of the mothers addressed, and an additional 200 replies were obtained through the Board of Health nurses, who went to the homes for the information.

Table 1 gives the results of this investigation — the number of replies received for babies of various ages and mother nativities and the percentages of breast- and bottle-fed babies.

TABLE 1.—LIVING BABIES—BREAST- OR BOTTLE-FED

Mother Nativity	2 Weeks to 1 Year	Total Replies												Percentage Breast Fed
		2 Weeks to 1 Year		2 Weeks to 1 Mo.		1 to 3 Months		3 to 6 Months		6 to 9 Months		9 to 12 Months		
		Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	
United States	272	174	98	44	8	33	23	42	19	24	29	31	19	64
Ireland	120	93	27	17	1	24	6	23	8	16	5	13	7	77
England, Scotland and Wales	20	15	5	1	..	3	1	4	1	3	2	4	1	..
Germany	2	1	1	..	..	..	..	1	1	..	..	..	..	..
Canada	69	38	31	5	1	8	7	8	9	8	8	9	6	55
Scandinavia	10	8	2	1	..	1	1	2	..	3	1	1	..	..
Italy	123	106	17	24	2	23	4	23	2	18	2	18	7	86
Russia and Poland	120	98	22	20	2	21	7	21	6	17	6	19	1	82
Totals	736	533	203	112	14	113	49	124	46	89	53	95	41	72
Percentages	...	72	28	89	11	70	30	73	27	63	37	70	30	..

The number of replies was 736, of which 533, or 72.4 per cent., were breast fed, and 203, or 27.6 per cent., were bottle-fed. But the information thus obtained was only for living children. To determine what proportion of all babies was bottle- or breast-fed, it was necessary to add a proportionate number of the deaths of 1911. These necessary additions are given in Table 2. Adding the figures in each square of Table 2 to the figures in the corresponding square of Table 1, totals may be obtained which show the relative proportions of breast- and bottle-fed babies in Boston. The corrected percentages shown in Table 2 were thus obtained. At various places in the table there appear higher percentages of breast feeding for the older babies than for the younger. This, of course, is an

error due to too few replies and would be rectified if answers from all the mothers of 1911 could be tabulated. But the evidence furnished by these 736 replies is very striking and may be used for present calculations.

It is estimated that 68 per cent. of all Boston babies between the ages of 2 weeks and 1 year are breast-fed; that of Boston babies with Canadian mothers only 51 per cent. are breast-fed; of those with native mothers 59 per cent.; of those with Irish mothers 73 per cent.; of those with Russian and Polish mothers 79 per cent., and of those with Italian mothers 83 per cent. The replies from mothers born in Scotland, England and Wales, Germany and Scandinavia were too few to warrant any estimates for their babies.

TABLE 2.—DEATH ADDITIONS TO TABLE 1. NECESSARY TO OBTAIN CORRECTED PERCENTAGES OF BREAST- AND BOTTLE-FED BABIES

Mother Nativity	2 Weeks to 1 Year		2 Weeks to 1 Mo.		1 to 3 Months		3 to 6 Months		6 to 9 Months		9 to 12 Months	
	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle
United States	5	27	1	3	1	9	1	7	1	4	1	4
Ireland	3	9	..	1	1	2	1	2	..	2	1	2
England, Scotland and Wales	1	1	..	..	..	..	1	1	..	..	..	..
Canada	2	7	..	1	1	2	1	2	..	2	..	..
Scandinavia	..	1	..	..	..	1	..	..	..	..	..	..
Italy	6	6	1	1	1	1	1	1	2	2	1	1
Russia and Poland	3	5	1	..	1	1	..	2	1	1	..	1
Totals	20	56	3	6	5	16	5	15	4	11	3	8
Percentages	68	32	85	15	64	36	68	32	59	41	67	33

CORRECTED PERCENTAGES OF BREAST-FED INFANTS OF VARIOUS MOTHER NATIVITIES

Children of mothers born in the United States	59
Children of mothers born in Ireland	73
Children of mothers born in Canada	51
Children of mothers born in Italy	83
Children of mothers born in Russia and Poland	79

Table 3 gives for 1911 the total mortality of infants by age, by mother nativity and by feeding (for infants over 2 weeks of age). Seventy-four per cent. of the deaths of these infants were among bottle-fed babies, and those between the ages of 1 and 3 months show the highest mortality, especially those having mothers born in the United States.

TABLE 3.—1911 MORTALITY OF INFANTS BY AGE, BY MOTHER NATIVITY AND BY FEEDING (FOR INFANTS OVER TWO WEEKS OF AGE)

Mother Nativity	Total Deaths*													Per Cent. of Deaths 2 Wks. to 1 Year, Breast-Fed.
	Under 1 Year	2 Weeks to 1 Year		2 Weeks to 1 Mo.		1 to 3 Months		3 to 6 Months		6 to 9 Months		9 to 12 Months		
		Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	
United States	928	106	546	26	58	20	172	31	145	13	93	16	78	16
Ireland	346	48	179	7	20	16	47	8	53	8	29	9	30	21
England, Scotland and Wales	61	13	25	1	1	4	7	5	10	1	4	2	3	34
Germany	12	3	6	1	..	..	2	..	1	1	..	1	3	33
Canada	232	32	128	5	10	7	40	7	33	7	35	6	10	20
Scandinavia	30	5	15	..	3	2	4	3	4	..	3	..	1	25
Italy	293	124	113	13	13	26	22	21	27	37	32	27	19	52
Russia and Poland	230	60	101	11	8	17	21	11	27	13	25	8	20	37
Other countries	87	21	51	..	1	6	12	5	10	6	18	4	10	29
Unknown	29	1	25	..	1	..	13	1	3	..	3	..	5	4
Totals	2,248	413	1,189	64	115	98	340	92	313	86	242	73	179	26
Percentages	.....	26	74	36	64	22	78	23	77	26	74	29	71	..

*There were three deaths over two weeks where method of feeding was not known.

TABLE 4.—1911 MORTALITY OF INFANTS BY MONTH, BY AGE AND BY FEEDING (FOR INFANTS OVER TWO WEEKS OF AGE)

Month	Total Deaths											
	Under 1 Year	2 Weeks to 1 Year		2 Weeks to 1 Mo.		1 to 3 Months		3 to 6 Months		6 to 9 Months		9 to 12 Months	
		Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle	Breast	Bottle
January	162	34	65	7	6	10	21	8	18	5	11	4	9
February	175	49	65	8	5	13	17	7	16	13	13	8	14
March	196	41	101	10	8	11	35	7	22	7	27	6	9
April	191	38	102	8	10	9	28	10	23	7	22	4	19
May	181	34	85	7	15	4	22	11	20	5	13	7	15
June	129	21	61	4	8	6	19	3	10	6	14	2	10
July	280	39	184	3	10	8	46	10	46	10	49	8	33
August	260	40	181	5	13	6	41	12	64	8	38	9	25
September	225	40	138	2	16	7	39	10	40	10	23	11	20
October	172	26	86	1	5	9	27	6	28	3	13	7	13
November	139	24	62	3	9	9	24	3	12	5	10	4	7
December	138	27	59	6	10	6	21	5	14	7	9	3	5
Totals	2,248	413	1,189	64	115	98	340	92	313	86	242	73	179

Now if 74 per cent. of infant deaths above the age of 2 weeks are among bottle-fed babies and only 32 per cent. of babies over 2 weeks are bottle-fed, then of such infants the bottle-fed is six times as likely to die as the breast-fed.

Table 4 gives for 1911 the mortality of infants by month, by age and by feeding (for infants over 2 weeks of age). During July and August the number of deaths among the breast-fed infants was only slightly increased, while among the bottle-fed infants the mortality was trebled.

An analysis of these deaths by cause reveals many interesting facts:

Measles.—Had a total of 17 deaths between 2 weeks and 1 year, 9 breast-fed and 8 bottle-fed, 6 of the breast-fed children were Italians, among whom measles always has a high mortality.

Whooping-Cough.—Had a total of 52 deaths, 23 breast-fed and 29 bottle-fed.

Using the deaths among the breast-fed children of each nationality as the standard, there were 40 per cent. too many deaths among the children of both native mothers and Irish mothers, and 35 per cent. too many deaths in the entire city.

Diphtheria.—Had a total of 20 deaths; all except 3, Italians, bottle-fed.

Pulmonary Tuberculosis.—Had a total of 16 deaths with only 1 breast-fed.

These figures regarding deaths from communicable diseases indicate that the bottle-fed baby either is more susceptible to these diseases or does not possess as much stamina as the breast-fed baby effectually to combat them. The same fact is apparent also in septicemia, which caused 5 deaths, all bottle-fed, and in diseases of the ear, which caused 6 deaths, all bottle-fed.

Bronchopneumonia and pneumonia had a total of 309 deaths between 2 weeks and 1 year with 184 bottle-fed. Using the deaths of breast-fed babies as the standard, breast feeding would have diminished these deaths above 2 weeks of age.

Among the children of native mothers 51 per cent.; Irish mothers, 53 per cent.; Canadian mothers, 43 per cent.; Italian mothers, 28 per cent.; Russian and Polish mothers, 37 per cent.; for the whole city, 40 per cent.

But it is especially in the study of diarrhea and enteritis that the terrible effects of bottle-feeding are revealed. There were in Boston in 1911, in infants between the ages of 2 weeks and 1 year, 621 deaths due to this cause, 534 of which were bottle-fed. From this cause the ratio of deaths of bottle-fed to breast-fed babies was, in the age period:

2 weeks to 1 month	4 to 1
1 to 3 months	9 to 1
3 to 6 months	7 to 1
6 to 9 months	8 to 1
9 to 12 months	3 to 1
All children between 2 weeks and 1 year	6 to 1
Those of native mothers	13 to 1
Irish mothers	7 to 1
Canadian mothers	12 to 1
Russian and Polish mothers	3 to 1
Italian mothers	3 to 2

TABLE 5.—INFANT MORTALITY-RATES FOR 1911 (USING BIRTHS OF 1910)

	Deaths per 1,000 Births	Deaths Per 1,000 Aged 2 Weeks to 1 Year.	Breast-Fed Children Aged 2 Weeks to 1 Year. Death Rates per 1,000 of Each Mother Nativity						Bottle-Fed Children Aged 2 Weeks to 1 Year Death Rates per 1,000 of Each Mother Nativity					
			All Mother Nativities.	American	Irish	Canadian	Italian	Russian and Polish.	All Mother Nativities.	American	Irish	Canadian	Italian	Russian and Polish
All causes	127	94	36	30	24	37	56	31	218	224	246	156	251	200
Communicable diseases (Nos. 1, 6, 7, 8, 9, 28-35, 61*a*, 61*b*, International Classification)	9	9	4	4	5	6	6	2	19	16	26	16	18	24
Broncho-pneumonia	11	11	6	5	4	4	14	4	21	21	25	10	38	16
Pneumonia	7	7	5	4	4	4	7	5	13	9	11	9	29	20
Diarrhea and enteritis	35	36	8	5	6	6	14	8	98	105	104	73	104	93
Congenital debility, icterus and sclerema and other causes peculiar to early infancy (Nos. 151 and 152, International Classification)	36	9	3	3	2	2	3	3	22	21	30	18	22	22

Perhaps the rates in Table 5 show the comparisons most clearly:

Of each 1,000 breast-fed infants over 2 weeks old of each mother nativity, there died

Americans 30
Irish 24
Canadians 37
Italians 56
Russians and Poles 31

while of each 1,000 bottle fed infants over 2 weeks old of each mother nativity there died

Americans 224
Irish 246
Canadians 156
Italians 251
Russians and Poles 200

and of each 1,000 breast-fed infants over 2 weeks old of each mother nativity, diarrhea and enteritis killed

Americans 5
Irish 6
Canadians 6
Italians 14
Russians and Poles 8

while of each 1,000 bottle-fed infants over 2 weeks of each mother nativity, diarrhea and enteritis killed

Americans 105
Irish 104
Canadians 73
Italians 104
Russians and Poles 93

These estimated rates measure the possible reduction in infant mortality if all babies could be breast-fed.

Of infants reaching the age of 2 weeks, one in five dies before a year old if bottle-fed, while if breast-fed only one in thirty fails to reach the 1 year mark. This means that the deaths of these infants would be 60 per cent. less if all could be breast-fed.

The actual number of infants deaths in Boston last year was 2,248. Breast feeding would have saved nearly a thousand of these children and the rate instead of 127 per thousand births would have been 71.

Such a saving of infant life is not a mere fancy. The reduction of the infant mortality rate from 127 to 71 is entirely within the range of possibilities.

New Zealand, in 1909, had a rate of 62 and South Australia for the same year a rate of 61.[10]

Surely mothers should suckle their children as God intended.

TABLE 6.—ACTUAL DEATH-RATES AND ESTIMATED DEATH-RATES IF ALL CHILDREN HAD BEEN BREAST-FED

	Mother Nativity					
	United States	Ireland	Canada	Italy	Russia and Poland	Totals for Boston
Births, 1910	6,226	2,816	1,747	2,705	2,479	17,670
Deaths under one year, 1911	928	346	232	293	230	2,248
Rate per 1,000 births (using the 1910 births)	149	123	133	108	93	127
Percentage of children between two weeks and one year breast-fed (estimated)	59	73	51	83	79	68
Deaths, 1911, of children between two weeks and one year	654	227	160	237	162	1,605
Percentage of deaths between two weeks and one year breast-fed	16	21	20	52	37	26
Estimated total infant deaths, 1911, if all above age of two weeks had been breast-fed	455	185	135	206	145	1,253
Lives saved if all above age of two weeks had been breast-fed	473	161	97	87	85	995
Estimated death rate if all above two weeks had been breast-fed	73	66	77	76	58	71
Estimated reduction in death-rate if all above two weeks had been breast-fed	76	57	56	32	35	56

10. Victorian Year Book, 1909-10, p. 132.

TABLE 7.—BOSTON 1911 MORTALITY OF INFANTS BY CAUSE, BY SEX, BY AGE, BY MOTHER NATIVITY,

Cause of Death	Total Deaths Under 1 Year		Two Weeks to One Year				2 Weeks to 1 Month		1 to 3 Months		3 to 6 Months		6 to 9 Months		9 to 12 Months		United States		Ireland		England, Scotland and Wales		Germany		Canada	
			Male		Female																					
	Male	Female	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.
All causes	1263	985	225	659	188	528	64	115	98	340	92	313	86	242	73	179	106	546	48	179	13	25	3	6	32	128
6. Measles	5	12	3	2	6	6	..	...	..	...	..	2	3	2	6	4	1	5	1	2	..	..	..	1	..	...
7. Scarlet fever	2	...	...	2	...	...	..	...	..	...	..	...	..	1	..	1	...	...	...	...	..	..	..	..	..	1
8. Whooping-cough	23	29	12	11	11	18	3	...	6	6	7	9	1	6	6	8	7	13	4	6	1	2	..	1	3	3
9. Diphtheria and croup	9	11	2	7	1	10	..	1	..	6	..	1	1	2	2	7	...	4	..	5	..	..	..	..	..	2
10. Influenza	1	1	1	...	1	...	1	...	...	...	...	...	1	...	...	...	...	...	1	...	..	..	..	..	1	...
14. Dysentery	5	4	3	2	2	2	..	...	1	...	1	1	2	3	1	...	1	3	..	...	..	..	1	..	1	...
18. Erysipelas	5	9	1	4	3	6	1	1	2	5	..	1	1	3	..	...	2	5	..	1	1	..	..	..	..	...
19. Other epidemic diseases	3	...	2	1	...	...	1	...	..	1	1	...	..	...	..	...	1	1	..	...	..	..	..	..	..	...
20. Purulent infection and septicemia	5	...	...	5	...	...	..	1	..	1	..	2	..	1	..	...	...	1	..	1	..	1	..	..	..	2
24. Tetanus	1	...	1	...	...	...	1	...	..	...	..	...	..	...	..	...	...	...	..	...	..	..	..	..	..	...
28. Tuberculosis of Lungs	7	5	...	7	1	4	..	...	..	1	..	3	1	4	..	3	...	8	1	...	..	..	..	..	..	1
29. Acute miliary tuberculosis	2	3	...	2	...	3	..	...	..	...	..	1	..	3	..	1	...	1	..	3	..	..	..	..	..	...
30. Tuberculous meningitis	21	11	9	11	3	8	..	...	2	2	5	3	3	6	2	8	5	7	3	2	..	..	..	..	1	3
31. Abdominal tuberculosis	3	2	...	3	...	2	..	1	..	1	..	...	..	1	..	2	...	1	..	1	..	..	..	..	..	2
34. Tuberculosis of other organs	1	...	...	1	...	...	..	...	..	...	..	...	..	1	..	...	...	...	..	...	..	..	..	..	..	1
36. Rickets	1	...	...	1	...	...	..	...	..	...	..	...	..	...	..	1	...	1	..	...	..	..	..	..	..	...
37. Syphilis	23	17	6	13	4	11	1	3	4	10	5	6	..	5	..	...	3	9	..	3	1	1	..	..	1	3
38. Gonococcus infection	2	1	...	2	...	1	..	1	..	2	..	...	..	...	..	...	...	2	..	...	..	..	..	..	..	1
45. Cancer and other mal. tumors of other organs	....	1	...	...	1	...	..	...	..	...	..	...	..	...	1	...	1	...	..	...	..	..	..	..	..	...
47. Acute articular rheumatism	1	...	...	1	...	...	..	...	..	...	..	...	..	1	..	...	...	1	..	...	..	..	..	..	..	...
49. Scurvy	1	...	...	1	...	...	..	...	..	...	..	...	..	...	..	1	...	1	..	...	..	..	..	..	..	...
54. Anemia chlorosis	1	...	...	...	...	...	..	...	..	...	..	...	..	...	..	...	...	...	..	...	..	..	..	..	..	...
55. Other general diseases	3	2	...	2	...	...	..	...	..	1	..	1	..	...	..	...	...	1	..	...	..	..	..	..	..	1
60. Encephalitis	1	...	1	...	...	...	..	...	..	...	..	...	1	...	..	...	...	...	..	...	..	..	..	..	..	...
61. Simple Meningitis	12	11	3	8	3	8	..	2	..	2	2	4	4	4	..	4	...	9	1	5	..	..	..	..	2	2
61a Cerebrospinal Meningitis (undefined)	2	1	1	1	1	...	..	...	1	...	..	1	1	...	..	...	1	...	..	...	..	..	..	..	1	...
61b Cerebrospinal fever	2	1	...	2	...	1	..	...	..	...	..	...	..	1	..	2	...	1	..	...	..	..	..	..	..	...
64. Cerebral hemorrhage apoplexy	2	2	...	...	...	...	..	...	..	...	..	...	..	...	..	...	...	...	..	...	..	..	..	..	..	...
71. Convulsions of infants	10	8	3	6	1	4	2	1	1	2	..	2	1	1	..	4	...	4	..	2	..	..	..	..	..	2
73. Neuralgia and neuritis	1	...	...	1	...	...	..	...	..	...	..	...	..	1	..	...	...	...	..	1	..	..	..	..	..	...

BY MONTH AND BY FEEDING (FOR INFANTS OVER TWO WEEKS OF AGE)

Scandinavia		Italy		Russia and Poland		Other Countries		Unknown		January		February		March		April		May		June		July		August		September		October		November		December	
Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.
5	15	124	113	60	101	21	51	1	25	34	65	49	65	41	101	38	102	34	85	21	61	39	184	40	181	40	138	26	86	24	62	27	59
...	...	6	...	...	...	1	..	..	..	1	..	3	..	..	...	2	...	2	2	1	..	..	1	..	...	..	...	..	2	..	3	..	..
...	...	...	...	...	1	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	1	..	...	..	...	..	...	..	...	..	..	..	1
1	...	3	1	3	2	1	1	..	..	1	3	4	4	1	7	2	2	4	3	3	..	1	2	3	1	..	3	1	2	2	1	1	1
...	...	3	3	...	2	..	..	..	1	..	..	..	1	..	1	..	3	1	4	..	1	..	6	..	...	1	1	1	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	1	...	1	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	1	2	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	3	1	1	1	..	2	..	..	1	..
...	...	1	2	...	1	..	1	..	..	1	..	..	1	1	4	..	1	..	2	..	1	..	...	1	...	..	1	..	..	..	..	1	..
...	...	...	...	1	...	..	..	..	..	1	..	..	..	..	...	..	1	..	..	..	..	1	..	..	...	..	..	..	..	..	..	..	..
...	...	...	...	1	...	..	..	..	..	..	..	..	..	1	1	..	..	..	1	..	..	..	1	..	...	..	1	..	1	..	..	..	..
...	...	...	...	1	...	..	..	..	..	..	..	..	..	1	...	..	..	..	..	..	..	..	..	..	...	..	..	..	..	..	..	..	..
...	...	...	1	...	1	..	..	..	..	..	1	..	3	1	2	..	1	..	..	..	..	..	1	..	...	..	...	..	3	..	..	..	..
...	...	...	...	...	...	..	1	..	..	..	1	..	..	..	...	..	...	..	2	..	..	..	...	..	...	..	1	..	1	..	..	..	..
...	1	1	3	1	3	..	..	1	..	2	4	4	2	1	1	2	...	..	4	..	1	..	3	3	1	..	1	..	1	..	..	..	1
...	...	...	...	...	...	..	..	..	1	..	..	..	..	..	...	..	...	..	..	..	1	..	1	..	...	..	2	..	..	..	1	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	1	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	...	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	1	..	...	..	...	..	..	..	..
...	...	4	3	1	...	..	2	..	3	..	1	1	1	2	1	..	...	..	1	1	1	1	5	1	3	..	2	2	4	1	2	1	3
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	3	..	...	..	..	..	..	..	...	..	...	..	...	..	...	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	1	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	...	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	1	..	...	..	..	..	..	..	...	..	...	..	...	..	...	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	...	..	1	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	...	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	1	..	...	..	...	..	1	..	..	..	...	..	...	..	...	..	...	..	..	..	..
...	...	1	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	...	1	..	..	..
...	...	3	...	...	...	..	..	..	..	..	3	1	..	2	...	..	3	..	3	..	..	2	4	..	...	..	1	..	1	..	..	1	1
...	...	...	...	...	1	..	..	..	..	..	..	1	..	..	...	..	...	..	..	..	..	1	...	..	...	..	...	..	1	..	..	..	..
...	...	...	...	...	2	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	1	..	1	..	...	..	...	..	1	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	...	..	..	..	..
...	...	1	2	2	...	1	..	..	..	1	1	..	2	1	2	2	1	..	1	..	..	..	1	..	1	..	...	..	...	..	1	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	1	..	..	..	..	..	...	..	...	..	...	..	...	..	..	..	..

TABLE 7.—BOSTON 1911 MORTALITY OF INFANTS BY CAUSE, BY SEX, BY AGE, BY MOTHER NATIVITY,

Cause of Death	Total Deaths Under 1 Year		Two Weeks to One Year				2 Weeks to 1 to 3		1 to 3 Months		3 to 6 Months		6 to 9 Months		9 to 12 Months		United States		Ireland		England, Scotland and Wales		Germany		Canada	
			Male		Female																					
	Male	Female	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.
74. Other diseases of nervous system.	1	5	...	1	...	5	..	1	..	1	..	1	..	2	..	1	...	5	..	...	..	..	..	..	..	...
75. Diseases of eyes and their annexa	2	...	...	2	...	...	..	...	..	2	..	...	..	...	..	...	...	1	..	...	..	..	..	..	..	...
76. Diseases of ears.	2	4	...	2	...	4	..	...	..	...	..	1	..	2	..	3	...	3	..	...	..	..	..	..	..	...
78. Acute endocarditis	....	2	...	...	...	2	..	...	..	1	..	...	..	1	..	...	...	...	..	...	..	2	..	..	..	...
79. Organic diseases of heart	....	3	...	...	1	2	..	...	..	...	..	1	1	...	..	1	...	...	..	...	..	..	..	..	1	1
82. Thrombosis and embolism	....	1	...	...	...	...	..	...	..	...	..	...	..	...	..	...	...	...	..	...	..	..	..	..	..	...
84. Diseases of lymphatic system	13	6	4	7	...	5	..	...	2	4	..	3	1	3	1	2	2	10	..	...	..	..	..	..	..	...
87. Diseases of larynx	....	2	...	...	1	1	..	...	1	...	..	...	..	...	..	1	...	...	..	...	..	..	..	..	..	...
89. Acute bronchitis.	24	18	12	8	7	8	3	3	7	4	4	5	5	2	..	2	2	6	2	2	..	..	..	..	1	2
90. Chronic bronchitis	....	2	...	...	...	2	..	...	..	...	..	...	..	2	..	...	...	1	..	...	..	..	..	..	..	...
91. Bronchopneumonia	106	84	38	64	31	51	7	6	16	30	14	27	20	30	12	22	17	52	7	18	2	2	..	..	3	8
92. Pneumonia	75	56	26	44	30	25	4	2	9	5	12	16	14	23	17	23	13	23	7	8	4	1	1	1	3	7
93. Pleurisy	1	...	...	1	...	...	..	...	..	...	..	...	..	1	..	...	...	...	..	...	..	..	..	..	..	...
94. Pulmonary congestion, pulmonary apoplexy.	4	1	...	4	...	1	..	1	..	...	..	2	..	...	..	2	...	3	..	...	..	..	..	..	..	...
100. Diseases of pharynx	....	2	...	...	1	1	..	...	..	...	1	...	..	1	..	...	1	...	..	...	..	..	..	..	..	1
103. Other diseases of stomach (except cancer) ..	5	2	2	2	1	...	2	...	..	1	1	1	..	...	..	...	1	2	..	...	..	..	..	..	..	...
104. Diarrhea and enteritis under 2 years	332	295	43	286	44	248	7	31	19	172	24	168	14	105	23	58	19	255	11	76	..	9	..	3	5	60
108. Appendicitis and typhilitis	....	1	...	...	...	...	..	...	..	...	..	...	..	...	..	...	...	...	..	...	..	..	..	..	..	...
109. Hernia, intestinal obstruction	17	10	9	5	3	4	2	1	2	5	3	2	4	1	1	...	2	8	1	...	..	..	..	..	4	1
110. Other diseases of intestines	1	...	...	...	...	...	..	...	..	...	..	...	..	...	..	...	...	...	..	...	..	..	..	..	..	...
115. Other diseases of liver	1	1	1	...	...	...	..	...	..	...	1	...	..	...	..	...	...	...	..	...	1	..	..	..	..	...
117. Simple peritonitis (non puerperal)	4	1	2	1	1	...	1	...	2	...	..	1	..	...	..	...	1	...	1	...	..	..	..	..	..	1
119. Acute nephritis .	5	1	1	3	...	1	1	...	..	2	..	...	..	1	..	1	1	1	..	2	..	..	..	..	..	...
120. Bright's Disease.	2	1	1	1	...	1	..	...	..	...	1	1	..	1	..	...	1	1	..	1	..	..	..	..	..	...
122 Other diseases of kidney and annexa	5	1	...	5	...	1	..	...	..	2	..	2	..	...	..	2	...	2	..	1	..	..	..	..	..	1

BY MONTH AND BY FEEDING (FOR INFANTS OVER TWO WEEKS OF AGE)—Continued

Scandinavia		Italy		Russia and Poland		Other Countries		Unknown		January		February		March		April		May		June		July		August		September		October		November		December	
Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.
...	...	...	...	...	1	..	..	..	..	..	..	..	..	..	2	..	...	..	..	..	1	..	...	..	...	..	...	..	1	..	1	..	1
...	...	...	...	...	...	..	1	..	..	..	..	..	..	..	...	..	...	..	1	..	1	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	...	..	3	..	..	..	1	..	1	..	...	..	3	..	..	..	..	..	...	..	...	..	...	..	..	..	1	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	1	..	1	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	1	..	..	..	..	..	..	1	..	..	1	..	...	..	..	..	..	..	1	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	1	2	1	..	..	..	..	..	..	1	..	2	...	..	1	..	..	..	2	..	2	..	2	..	...	..	1	..	1	1	3
...	...	1	..	..	...	..	1	..	..	..	..	..	..	..	...	..	...	..	..	1	1	..	...	..	...	..	...	..	..	..	..	..	..
1	2	10	5	2	...	1	1	..	..	1	2	5	..	3	5	4	1	1	..	1	1	..	1	..	1	..	3	1	..	2	2	1	..
...	...	...	...	...	...	..	1	..	..	..	..	..	..	..	...	..	...	..	1	..	..	..	...	..	...	..	1	..	..	..	..	..	..
3	8	3	30	17	8	8	2	5	..	2	9	5	5	15	9	12	6	13	8	7	4	6	5	11	8	7	2	8	2	7	5	10	6	14
3	7	2	16	13	9	10	3	3	..	1	4	4	11	6	5	4	5	14	8	9	3	5	3	8	2	1	4	6	3	3	3	3	5	6
...	...	...	...	...	1	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	1	..	...	..	...	..	..	..	..	..	..	..
...	...	...	...	...	...	..	1	..	1	..	2	..	..	..	1	..	1	..	..	..	..	..	...	..	...	..	...	..	1	..	..	..	..	..
1	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	1	1	..	..	..	...	..	...	..	...	..	..	..	..	..	..	..
...	...	1	...	1	...	..	..	..	..	1	..	..	1	1	...	..	...	..	1	..	..	..	...	..	...	..	...	..	..	..	..	..	1	..
60	...	2	30	47	15	47	7	21	..	14	3	17	2	9	..	28	6	40	2	25	1	24	14	99	16	146	27	80	10	39	3	17	3	10
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..	..
1	...	2	...	2	...	1	..	..	..	2	4	..	..	3	1	1	1	2	..	1	..	1	1	1	1	1	..	...	..	..	1	1	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	1	..	..	..	..	..
1	1	...	...	...	...	..	..	..	..	..	..	..	..	..	...	2	...	..	..	1	..	..	1	..	...	..	...	..	..	..	..	..	..	..
...	...	...	...	...	...	..	1	..	..	1	1	..	..	..	1	..	...	..	..	..	1	..	...	..	1	..	...	..	..	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	1	1	..	1	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..	..
1	...	...	1	...	...	..	..	..	1	..	..	..	..	..	...	..	1	..	..	..	..	..	1	..	2	..	1	..	..	..	..	1	..	..

TABLE 7.—BOSTON 1911 MORTALITY OF INFANTS BY CAUSE, BY SEX, BY AGE, BY MOTHER NATIVITY,

Cause of Death	Total Deaths Under 1 Year		Two Weeks to One Year				2 Weeks to 1 Month		1 to 3 Months		3 to 6 Months		6 to 9 Months		9 to 12 Months		United States		Ireland		England, Scotland and Wales		Germany		Canada	
			Male		Female																					
	Male	Female	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.
144. Acute abscess ...	3	3	...	3	...	3	..	...	..	2	..	1	..	2	..	1	...	6	..	...	..	..	..	..	..	...
145. Other diseases of skin and annexa	11	2	1	9	1	1	2	3	..	...	..	5	..	...	..	2	2	5	..	1	..	..	..	..	..	2
146. Diseases of bone (tuberculosis excepted)	4	4	1	3	1	3	..	...	2	...	..	3	..	1	..	2	...	3	..	...	..	..	..	..	1	1
150. Congenital malformations (still-birth excepted)	79	61	9	22	10	17	6	9	5	16	3	6	5	6	..	2	6	13	3	11	..	2	..	..	2	3
151. Congenital debility, icterus and sclerema	264	192	20	61	9	44	16	39	10	42	2	16	1	5	..	3	10	45	4	20	..	3	1	..	2	13
152. Other diseases peculiar to early infancy .	119	69	2	10	1	4	3	7	..	7	..	...	..	...	..	...	1	7	..	2	..	..	..	..	..	2
153. Lack of care ...	1	1	...	1	1	...	..	...	1	...	..	1	..	...	..	...	...	...	..	1	1	..	..	..	..	...
167. Burns (conflagration excepted)	2	1	1	...	...	...	..	...	1	...	..	...	..	...	..	...	...	...	1	...	..	..	..	..	..	...
169. Accidental drowning	....	1	...	...	...	...	..	...	...	...	..	...	..	...	..	...	...	...	..	...	..	..	..	..	..	...
172. Traumatism by fall	1	...	...	1	...	...	..	...	..	...	..	1	..	...	..	...	...	...	..	...	..	..	..	..	..	...
177. Starvation	....	1	...	...	...	1	..	...	..	...	..	1	..	...	..	...	...	1	..	...	..	..	..	..	..	...
179. Effects of heat..	8	3	...	8	2	1	..	...	..	2	..	3	1	2	1	2	1	6	..	1	..	..	..	..	..	1
184. Homicide by other means ...	1	...	...	...	...	...	..	...	..	...	..	...	..	...	..	...	...	...	..	...	..	..	..	..	..	...
186. Other external violence	2	1	2	...	1	...	..	...	3	...	..	...	..	...	..	...	1	...	..	...	..	..	..	..	..	...
187. Ill defined organic disease ..	1	...	...	1	...	...	..	...	..	...	..	1	..	...	..	...	...	1	..	...	..	..	..	..	..	...
188. Sudden death ...	1	1	...	1	1	...	..	...	..	...	1	1	..	...	..	...	...	1	..	...	..	..	..	..	..	...
189. Cause of death not specified or ill defined	11	14	2	8	3	9	..	1	1	2	4	6	..	5	..	3	3	7	..	3	2	2	..	..	..	...

BY MONTH AND BY FEEDING (FOR INFANTS OVER TWO WEEKS OF AGE)—Continued

Scandinavia		Italy		Russia and Poland		Other Countries		Unknown		January		February		March		April		May		June		July		August		September		October		November		December	
Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.	Br.	Bot.
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	1	..	1	..	2	..	1	..	...	..	...	..	..	..	1	..	..
...	1	...	...	...	1	..	..	..	..	..	..	1	1	1	2	..	2	..	3	..	1	..	...	..	...	..	...	..	1	..	..	..	..
...	...	...	1	1	...	..	1	..	..	1	4	..	..	..	...	..	...	..	..	..	1	1	1	..	...	..	...	..	..	..	..	..	..
1	2	3	2	4	4	..	2	..	..	1	2	2	3	1	3	1	2	1	..	3	2	4	8	..	2	3	4	1	4	2	5	..	4
1	2	5	10	4	10	2	2	..	..	4	6	3	8	4	10	2	7	3	8	..	6	1	9	2	6	2	17	3	10	..	6	5	12
...	2	1	...	1	1	..	..	..	..	..	1	..	1	..	4	..	...	..	2	1	1	..	1	..	1	..	1	..	1	2	..	..	1
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	1	...	..	1	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	1	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	...	..	1	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	1	..	..	..	..	..	..
...	...	...	...	1	...	..	1	..	..	..	..	..	..	..	...	..	...	..	..	..	..	2	9	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	1	...	1	...	..	..	..	..	..	..	1	..	1	...	..	...	1	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	...	...	...	...	..	..	..	..	..	..	..	1	..	...	..	...	..	..	..	..	..	...	..	...	..	...	..	..	..	..	..	..
...	...	1	...	...	...	..	..	..	..	..	..	..	..	..	...	..	...	..	..	..	..	..	...	..	...	..	1	..	..	1	..	..	..
...	...	...	1	...	2	..	1	..	1	..	2	..	2	..	3	1	1	..	..	..	..	2	3	..	2	..	1	1	..	1	2	..	1

VULVOVAGINITIS IN CHILDREN

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For two years and a half, under the direction of Dr. William P. Lucas, the cases of vaginitis at the Children's Hospital in Boston have been treated at a special clinic. During the two years the individual cases have been studied and the attempt has been made to gain some progress in the treatment and knowledge of this very intractable disease.

The purpose of this article is to emphasize the following five things, which stand out clearly as the result of our experience and the experience of others:

1. The prevalence of the gonococcus as the etiologic factor in cases of vaginitis, and the unreliability of bacteriologic examination in all stages.

2. The total duration of the disease, including the long periods of latency.

3. The importance of the disease on account of its serious complications and sequelae.

4. Inefficiency of treatment at best.

5. The consequent importance of prophylaxis, both at home and in hospitals.

ETIOLOGY

1. It is extremely interesting to read the history of vaginitis in children and see how opinions concerning its etiology have fluctuated during the last two centuries. Epstein[1] gives us a very interesting and complete account of it in the *Traité dès Maladies de l'Enfance,* an extremely valuable treatise and one not often mentioned in bibliographies on the subject. Alice Hamilton[2] has also given us, in English, an interesting account of its history.

According to Epstein, it was mentioned in medical literature at the beginning of the eighteenth century, but was then considered a phenomenon similar to the precocious menstruation of infants. After the middle of the eighteenth century, influenced by Hunter's beliefs and experiments, it became identified with syphilis, and this confusion still continued, according to some authorities at least, until the middle of the nineteenth century. In the meantime (1821) a new theory had arisen, and besides

1. Epstein: Maladies dès l'Enfance, iii, Chap. xxvii.
2. Hamilton, Alice: Gonorrheal Vulvovaginitis in Children. Jour. Infect. Dis., March 30, 1908, v, 147.

many cases which were still thought to be due to syphilis, others were thought to be due to such causes as teething, digestive disturbances and a lymphatic diathesis. In 1835, Ricord separated the two diseases, but even then denied the contagiousness of the gonorrhea. It was then that the pendulum swung to the other extreme and the vaginal discharge of infants was considered for the next twenty-five years or so as a disease produced by many different irritants and associated with many diseases, but always of minor importance and never contagious or connected in any way with venereal infections. Behrund (1842) introduced the name vulvovaginitis and distinguished the following forms: catarrhal or erythematous, phlegmonous, gangrenous, diphtheritic, syphilitic and eruptive or exanthematous.

Gradually the clinical experience of the disease in adults began to have its influence on the conception of the disease in children, and it was acceded from 1860 to 1870 that acute leukorrhea in childhood might be gonorrhea in the cases in which there had been direct contact.

About this time epidemics began to be noted, and finally Pott of Halle, through his personal observations, came to believe that the vulvovaginitis of children was identical with the gonorrhea of adults. He and his contemporaries also concluded that the disease could be acquired innocently. Even after the discovery by Neisser, in 1879, of the diplococcus, there was, and perhaps still is, a great diversity of opinion as to how large a proportion of the cases of vulvovaginitis in children is due to gonococcus.

Most text-books divide it, however, into a simple vaginal catarrh and a specific vaginitis of gonococcal origin. (See Carr, Cautley, Chapin, Cotton, Holt, Kelley, Kerley, Rotch and Starr.)

According to Goodhart and Still, "All cases should be treated as infectious. It would seem that whether the gonococcus is present in the discharge or not, the disease may spread from one child to another, and also to adults."

Koplik[3] describes under non-specific catarrhal vaginitis three types:

1. Normal discharge in the new-born which lasts about two weeks.

2. A discharge resulting from uncleanliness, pin-worms and masturbation.

3. An infectious discharge communicable from one child to another which may last for months and again occur. The exact etiology is unknown; uncleanliness, infection from vaginal discharge, marasmus, the infectious diseases or frail health may be the cause. Microscopically, there are seen in the pus cells bacteria and diplococci in groups which do not show by culture or on staining the characteristics of the gonococcus.

3. Koplik: Diseases of Infancy and Childhood, 1910.

Langstein[4] (Pfaundler and Schlossmann) says regarding vulvovaginitis that it is only in the last two decades that we have come to realize its terrible frequency, but that the experience of the last few years has taught us that the great majority of cases are infectious and are due to the gonococcus. He mentions also under vulvovaginitis of other etiology, a catarrhal condition of the new-born, aphthous vulvitis, an impetiginous form due to the localization of impetigo contagiosa, and a vaginal catarrh resulting from herpes, from the localization of syphilitic lesions and from vaccine pustules. Besides these he speaks of a vulvitis occurring in the acute stage of small-pox, scarlet fever and measles, also a vulvovaginitis cachectica and scrofulosa, and finally a plegmon of the vulva and vagina which he considers a complication of the various other forms of inflammation.

Marfan[5] makes the following classification clinically:

1. Saprophytic vulvitis.

2. Pyodermic, impetiginous and aphthous vulvitis, due to staphylococci and streptococci, following chicken-pox and measles.

3. Pseudo-membranous vulvitis, caused by diphtheria.

4. Eczematous vulvitis of older girls which is non-specific and accompanied by extremely profuse discharge. In this condition the thighs are reddened and eczematous plaques are found on the abdomen. Marfan says further that the catarrhal condition of these last cases is extremely difficult to cure.

Considering the membranous condition of the vagina due to the Klebs-Loeffler bacillus as a manifestation of diphtheria, it seems to us that the conditions caused by the streptococcus, staphylococcus and diphtheroids may be classified as simple vaginal catarrh and are probably of very short duration and are non-communicable. This simplifies the classification into that which has been stated above and has already been adopted by many writers, i. e., simple vaginal catarrh and a specific vaginitis of gonococcal origin. We believe that all cases of vaginitis in which there is a profuse and persistent discharge are due primarily to the gonococcus, and that all epidemics may be proved to have the same origin.

Since our special clinic was started there have been eighty-three cases registered, fifty-six of which have shown the presence of gonococci on bacteriologic examination. Nine cases were distinctly non-gonococcal in character. In four there was slight irritation with no discharge, and in five there was a very slight discharge which lasted a week or two at the longest, and which disappeared after a few days' treatment. In one of these cases the bacterial examination showed an intracellular diplo-

4. Langstein: Diseases of Children. Edited by Pfaundler and Schlossmann.
5. Marfan: Vulvovaginite. Jour. de méd. de Paris, 1909, xx, 313.

coccus similar in shape to the Neisser organism, but which did not decolorize by Gram.

Three of the five cases of simple vaginal catarrh which we have seen were associated respectively with acute infectious diarrhea, pneumonia and with a "fall." These were all of very short duration, however, and do not correspond to the picture in the text-books. The cases which we have had associated with measles, pin-worms and diphtheria have all shown the presence of the gonococcus.

Nineteen cases could not be identified by bacteriologic examination. Sometimes a case came but once, and a satisfactory smear could not be obtained. Again, a child may have come in the chronic stage, or just after the discharge had stopped, and in either case it is not always possible to discover the organism. Vail and Lincoln[6] give some excellent charts showing the disappearance of the gonococcus from vulva, vagina and urethra while the discharge is still present, and at the same time state that the absence of vulvovaginal discharge by no means rules out the diagnosis of gonorrhea.

In some cases we have found the smear to be positive for many months after the discharge has stopped, especially in untreated cases, and in other cases a positive smear may be difficult to obtain even in the active stage.

The following case illustrates this last condition:

R. E., aged 4½ years, came to the clinic Jan. 19, 1911. She had been treated outside for a discharge which she had had since October, 1910. The physician who had treated her had made no bacteriologic examination, but told the mother it was probably gonorrhea. The smear made when she came to the clinic showed a great many pus cells but no organisms of any kind. Alice Hamilton[2] states that it is generally conceded that a discharge rich in leukocytes and poor in bacteria should be regarded as very suspicious of gonorrhea. Cultures made on blood-agar showed a pure culture of streptococcus. The case continued with the same tenacity of a gonococcus infection, but no gonococci could be found in repeated bacteriologic examinations, until the middle of May. In the meantime smears were also taken by a physician outside the hospital to whom she went on account of a slight conjunctivitis, in accordance with our directions in the clinic. He, also, contrary to his expectations, was unable to find the Neisser organism. On May 18, 1911, when the discharge had been absent for a month, but while there was still a slight stain visible on the clothing, the smear showed a Gram-negative intracellular diplococcus not in the distinct morphology seen in the early stages, but very similar to the picture seen as a case is clearing up or becoming chronic. These were again found on June 19, 1911, and not in other smears taken in the interim. The possibility arises of this being a fresh infection from other cases in the clinic, but as the case showed no exacerbation at any time during the treatment, it seems hardly probable. We feel confident that this case was of Neisser origin and it seems to us an additional proof of the unreliability of smears. Maier[7] says that bacteriologic examination often fails in those cases in which it is most needed. The only explanation we have to offer for the long absence of positive smears in

6. Vail and Lincoln: Comparative Results in the Treatment of Gonorrhea in Young Girls. Ill. Med. Jour., 1909, lxi, 191.

7. Maier: Vulvitis and Vulvovaginitis of Children. Therap. Gaz., 1910, xxxiv, 540.

the above case is that the focus of infection may have been higher up than usual—that it may have extended into the uterus—because the child complained of abdominal pain in the early morning for several months, which gradually ceased as the discharge diminished. However, there was no tenderness found at any time over the tubes and ovaries, and the pain was never localized in their vicinity, so that it is improbable, even if the infection extended into the uterus, that it went beyond.

Abdominal pain of a more or less indefinite nature is not an uncommon symptom, especially in the chronic cases, and suggests that the infection extends beyond the vagina. It has occurred in nine of our cases. In one case it always preceded a slight recurrence of the discharge. In another it was associated with vomiting. It is especially common among the girls from 10 to 12 years of age, and strongly suggests the further extension of the infection into the developing organs as puberty approaches.

The following case may be of interest as simulating the saprophytic vulvitis described by Marfan:

A. C. came to us March 6, 1911, with a very foul, purulent discharge. The previous summer there had been a discharge with foul odor which was not very profuse, but which lasted two months without treatment. Two months previously it had begun again according to the mother, although the child thought it had persisted since the previous summer. The smears showed organisms of all kinds, and among others, Gram-negative intracellular diplococci which, however, were smaller than the Neisser organism and not definitely biscuit shaped. There certainly was a putrefactive organism present in the discharge but whether the Neisser organism would have been found if smears could have been obtained early in the disease, or if the case could have been watched over a longer period, it is impossible to say. However, during six weeks, no positive smears could be obtained, although it was often very suspicious.

As the ordinary case clears up, it is the common experience to find a variety of organisms coming to take the place of the gonococcus and it has also happened that where a foreign substance has been left in the vagina and putrefaction has gone on, the gonococcus has disappeared. Either of these conditions may have explained the absence of the gonococcus in this case. Although we have no proof to offer, judging from the clinical picture of this case and the suspicious character of the smears, we believe that the Neisser organism was probably the initial infecting agent.

We have had one case which it has seemed difficult to classify, but which was under treatment for six months. At no time, however, had there been any profuse discharge or even enough to cause a stain on the clothing. The case suggested irritation from hyperacid urine, but the examination, as will be seen, was negative.

V. O., seen Jan. 30, 1911, was 11 years old. For three or four years there had been watery secretion from the vagina, which was not sufficient to make any stain on the clothing. For two months she had complained of itching of external genitalia, and pain and burning on urinating. Examination showed the hymen not intact and slight redness of vulva, with some cheesy secretion between the labia majora. There was practically no discharge from the vagina. Examination of the urine was as follows: Very pale in color, reaction neutral, specific gravity 1,010, no sugar or albumin. Sediment showed epithelial cells, mostly squamous

epithelium, and no pus cells. The smear showed a long, thick bacillus in pure culture, similar to that found in the normal vaginal mucosa of adults. There had been a history of masturbation two years previously, and parents were not sure it did not still exist.

The child was under treatment for six months for the pruritus rather than for any discharge. It made steady improvement and finally cleared up entirely under applications of a solution of silver nitrate to the irritated surfaces and instillations of argyrol into the vagina. This case we believe was at no time due to the gonococcus, as the discharge was never profuse. It seemed to us to be a case of vulvitis rather than of vaginitis.

In Wallace Hamilton's[8] report of the ages of his 344 cases, the youngest child was 3 weeks, and the eldest was 12½ years old. The average age was 5 1/10 years. One hundred and fifty-one of his cases, or 43 per cent., were under 5 years.

Our ages correspond very closely to this—the average of seventy-one cases was 5 4/10 years. Our youngest child was 7 months; our oldest 11 years. Twenty-eight of the seventy-one children, or 39 per cent,, were 5 years old, or under.

SOURCE OF INFECTION

We have not been very successful in tracing the source of infection even with the cooperation of interested and intelligent mothers. The following is the result of our inquiries:

	Number	Per cent.
Discharged from hospital	16	21
History of discharge in one of parents	26	35
History of contact	3	4
Not discovered	30	40

Among 344 cases Hamilton[8] finds the following:

	Per cent.
Discharged from hospitals, day nurseries or homes	26
With history of leukorrhea in mother	20
Cases having slept with another child who had a gonococcal infection	9
Cases in which no cause was given	45

Among his 344 cases there was but one case of rape. In eighty-four cases we have had but one case of rape, and that was a case in which there was no infection.

The question of infection in hospitals and schools has been covered very thoroughly in medical literature, and yet epidemics still occur, and many children each year leave hospitals with an infection acquired there which may seriously handicap them through life. Alice Hamilton[2] has tabulated all the epidemics on record, which number 1,962 cases. One of the most famous of these is that one described by Skutsch,[9] which occurred in Posen, in 1890, in which, during the single month of August, 236 cases were reported among school children.

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8. Hamilton, Wallace: Gonococcus Vulvovaginitis in Children. Jour. Am. Med. Assn., April 9, 1910, l, 1196; Am. Jour. Obst., 1910, lxi, 837.

9. Skutsch: Ueber Vulvovaginitis gonorrhoica bei kleinen Mädchen; Inaugural Dissertation. Jena, 1891.

Holt[10] has given us an account of the experiences of the Babies' Hospital with regard to hospital epidemics, and their success at last in freeing the hospital from infection.

Butler,[11] in reviewing the most important causes of hospital epidemics, proves that the laundry is not responsible, but believes that the following are the more important, if not the predominating causes, of spreading such infections in children's hospitals: Taking smears; changing children; taking temperature, not from the thermometers, but from the hands; bathing in a common bath tub, or on a common slab, by a nurse whose hands have been contaminated by handling infants with no perceptible discharge; using the same toilet; and also the non-enforcement of tight cloth protection for all female children.

Weill and Barjon[12] have traced one epidemic to a thermometer. Bendig[13] has traced another of fifteen among forty children definitely to bath tubs and towels. Alice Hamilton[2] thinks it is introduced into hospitals by children with a discharge which is apparently non-gonococcal.

Out of about eighty-five cases which we have had in the clinic it has been possible to trace twenty-six and learn something of their subsequent course. As may be seen by the accompanying table, all but two had a recurrence of the discharge after intervals varying from three weeks to six years, and one of these two had a vulvovaginal abscess after the discharge had ceased, strongly suggesting that the infection was still present. It has recently been stated by a specialist in genito-urinary diseases that the cure of vaginitis in children is simple and effective, and that the so-called recurrences are really reinfections from some unknown source, and that the important thing in treating such cases is to discover the source.

While this may be true in some instances, we do not believe it to be true in the majority of cases, and feel that there is much evidence to the contrary. That twenty-two out of twenty-six of our cases have had a second attack is certainly a striking evidence of the similarity of the course of all cases, and I think it will be possible to prove many instances where a fresh infection may be ruled out. In the first place, many of these cases were undoubtedly due to hospital infections. No other cases in the same families resulted from the first case, and as many of the

10. Holt: Gonococcus Infections in Children with Special Reference to Their Prevalence in Institutions and Means of Prevention. New York Med. Jour., March 18, 1905, lxxxi, 521, 589.

11. Butler: Gonorrheal Vulvovaginitis; Its Specific Treatment and Prophylaxis. Interstate Med. Jour., July, 1910, xvii, 510.

12. Weill and Barjon: Congrès français de Medicine int. de Lyon, 1894.

13. Bendig: Ueber eine Gonorrhöeendemie bei Schulkindern in einem Salbad; München. med. Wchnschr., 1909, lvi, 1846.

TABLE SHOWING DURATION AND RECURRENCE OF ATTACKS OF VULVOVAGINITIS IN CHILDREN

No.	Duration of First Attack	Duration of Infection Before Beginn'g Treatment	Number of Recurrences	Longest Interval Between Attacks	Longest Time Present	Time Elapsed Since Any Evidence of Infection Was Present	Total Duration of Disease	Remarks
1	5 mos.	2 mos.	6	1 mo.	5 mos.	.....	2 yrs. 7 mos.	Still present
2	6 mos.	2 mos.	1	21 mos.	6 mos.	.....	2½ yrs.	Still present
3	.....	7 mos.	1	1 year	1 year	6 mos.	2½ yrs.	Smear positive for one year
4	6 mos.	6 mos.	2	1 yr. 8 mos.	6 mos.	3 mos.	2¼ yrs.	.....
5	4 mos.	4 wks.	0	0	4 mos.	17 mos.	4 mos.	.....
6	8 mos.	1 wk.	1	1 mo.	8 mos.	15 mos.	10 mos.	Vaccine treatment only. Local treatment impossible at home.
7	3 mos.	.....	1	5 years	6 mos.	4 mos.	6 years	Began 5 years ago. Present attack 3 weeks. Second attack observed in clinic, lasted 7 mos. First not seen.
8	10 mos.	3 mos.	1	1 mo.	10 mos.	6 mos.	1 year	.....
9	2 mos.	.....	2	3 mos.	2 mos.	Still present	6 mos. +	Lasted 2 mos. 7 mos. ago.
10	6 mos.	.....	8	7½ mos.	6 mos.	.....	4 years	Began 2 years ago. Present attack 6 mos. Returns as soon as treatment is discontinued. Has always been present in rectum.
11	6 mos.	2 wks.	4	1 mo.	6 mos.	1 week	13 mos.	Had only two treatments.
12	1 mo.	2 wks.	0	0	1 mo.	1 year	1 mo.	.....
13	few weeks	2d attack 1 wk.	1	6 years	6 wks.	1 year	7 years	Second attack lasted one mo.
14	2½ weeks	12 days	1	4 mos.	2½ wks.	5 mos.	7 years	Cleared up in five days.
15	4 weeks	2 wks. 5 dys.	1	2 mos.	4 wks.	4 mos.	8 mos.	Cleared up in nine days.
16	6 mos.	6 mos.	2	?	6 mos.	6 mos.	1½ yrs.	Cleared up in three days.
17	2 mos.	3 mos.	*	3½ mos.	5 mos.	5 mos.	11 mos.	.....
18	3 mos.	3 wks.	2	2 mos.	3 mos.	2 mos.	1 year	.....
19	?	2 wks.	2	1 year	3 mos.	Died	2 years	.....
20	4 mos.	3 wks.	1	1 mo.	4 mos.	.....	7 mos.	Still present
21	1 mo.	2 wks.	1	5 mos.	1 mo.	10 days	6 mos.	.....
22	1 year	2 wks.	4	6 mos.	1 yr.	3 mos.	2 years	.....
23	2 mos.	9 mos.	1	6 mos.	4 mos.	.....	12 mos.	Still present. Had discharge for 2 months, 9 months ago. Returned 1 month ago.
24	5 mos.	2 mos.	2	2 mos.	5 mos.	.....	14 mos.	No discharge. Smear suspicious.
25	3 mos.	2 wks.	1	3 wks.	3 mos.	. .....	4 mos.	Still present.
26	2 mos.	1 wk.	2	6 wks.	2 mos.	.....	4½ mos.	Still present.

APPROXIMATE AVERAGES

	4 mos.	5 mos.	2	9 mos.	4 mos.	.....	1 year	.....
	2 weeks	2 wks.	..	3 wks.	3 wks.	.....	8 mos.	.....

*Several.

mothers strongly suspected the origin and in some cases were quite sure of it, it is improbable that they would have been reticent about other infections had they occurred. Numbers 14, 15 and 17 in the accompanying table were definitely due to a hospital infection, and with no other known source it seems fair to assume that after intervals of four months, two months and three and a half months, respectively, the second attack was a recurrence of the initial infection rather than one of different origin.

The following case is another argument in favor of recurrences:

J. H., a girl of 12, had been under observation in the clinic since April 13, 1910. She had had a discharge the previous September, but at the time of beginning treatment in the clinic had no discharge although bacteriologic examination showed the presence of gonococci. For seven months smears showed an occasional intracellular Gram-negative biscuit-shaped diplococcus and always many extracellular ones. In December the smear became negative and she was not seen until October, 1911, ten months later, when she came for examination as she wished to enter the hospital for an operation on the orthopedic side. At that time there was a slight discharge and the smear was strongly positive showing many typical intracellular organisms. Under treatment the discharge disappeared and three negative smears were obtained during November, a week apart. In January, two months later, she was taken into the hospital, but kept under special precautions in spite of the negative smears. There was no other case in the hospital at the time, but during her stay she suddenly developed a profuse discharge. Was this not probably a recurrence?

The third argument in favor of recurrences is that a positive smear may persist for a long time after the discharge has ceased, especially in untreated cases, and often in treated ones. This is well illustrated in the case just cited. If an organism can remain in the vaginal mucosa for many months, is it not probable that when the vitality of the individual is lowered, the infection may suddenly start up and cause symptoms in the same manner that they do on the tonsils or in the pharynx?

The fourth argument is that in several families where there have been sisters the discharge has returned in the patient alone. It seems probable that if there was a source of infection in the family the sister would be open to the infection as well as the patient. In the case of J. H., already given, there were two sisters who were both examined in the clinic with negative results. There had been no history of a discharge in either case. Taking these arguments into consideration, we believe that the majority of subsequent attacks in twenty-four out of our twenty-six cases were recurrences of the original infection rather than a reinfection from some outside source.

The average duration of the first attack in thirty-five of our cases was ten weeks, the shortest was two weeks, the longest one year. Many of the cases were untreated during the first attack, often a year after the

onset. It is these cases that make the average duration so high. For even if the discharge responds quickly to treatment the organisms seem by that time to have invaded the tissues to such an extent that the discharge will probably recur and the smears remain positive for a long time.

The accompanying table shows the length of time these same cases had gone on before coming to the clinic. None of our cases came until the discharge had already existed one week, and one (not mentioned in the table) had already had a discharge at intervals for four years. The average duration of the cases before treatment was five months and one week. It is this fact that makes treatment so discouraging, for we believe with others[13] that early treatment favors the prognosis.

Another point of interest in these cases is that while in all but two the discharge returned at least once, in some it returned as many as eight times, making the average for each case twice. Again, while the longest interval between attacks was in several cases one month, in another it is reported to be five years.

In discharging patients from the clinic and in answering the questions of anxious mothers who have not too much patience in keeping up treatment after the discharge has subsided, it is customary to have some arbitrary period after which a case is pronounced cured. In one clinic[14] recovery means a complete cessation of the discharge and at least three or four negative smears from the vaginal mucosa at intervals of three or four days.

In another clinic[8] a case is pronounced cured after six negative bacteriologic examinations, the first four, one week apart and the last two, two weeks apart, covering a period of two months.

Again, another clinic[15] pronounces a case cured after four negative smears and the absence of discharge for eleven days.

At a home,[16] of which we shall later speak more fully, a child is not dismissed until six successive smears taken a week apart are negative and the local condition is satisfactory, during which time the child receives no vaccine.

Still another writer[11] considers recovery to be negative smears and no discharge. The cases are dismissed after several negative smears. Hamilton and Cooke,[17] however, do not claim that a complete cure was effected

14. Butler and Long: The Vaccine Treatment of Gonorrheal Vulvovaginitis in Children, Jour. Am. Med. Assn., March 7, 1908, l, 744; Vaccine and Serum Treatment of Gonorrhea in Female Children; ibid. Oct. 17, 1908, li, 1301.

15. Churchill and Soper: Inoculation Treatment of Vulvovaginitis in Children. Jour. Am. Med. Assn., Oct. 17, 1908, li, 1298.

16. Seippel: Venereal Diseases in Children. Ill. Med. Jour., July, 1912, xxii, 50.

17. Hamilton and Cooke; Inoculation Treatment of Gonorrheal Vulvovaginitis in Children. Jour. Infect. Dis., March 30, 1908, v, 158.

in any of the cases they reported. Schweitzer[18] also states that a case is not cured when clinical symptoms subside.

The frequency of recurrence after intervals of four, six and eight months, and sometimes a year or a year and a half, should make us pause before we pronounce our cases cured. One case (No. 4 in table) which we were able to keep under observation for a year after the discharge stopped and the smears had become negative and which we felt very confident was cured, showed a positive smear and a discharge one year and eight months later. It has seemed impossible in this case to find any fresh source for the second attack, and we have obtained a negative smear from examination of the mother. Moreover, in this case also the second attack did not in the least resemble a fresh infection, but was slight and of short duration.

The average of the total duration of the disease in twenty-six cases which it was possible to trace, was one year and eight months, which, no matter what the cause, is not very encouraging. The case which is reported to have lasted one month (No. 12 in table), and which has not been present for a year, is the one, however, in which there is a history of a vulvovaginal abscess, after the discharge stopped. Number 5 had a total duration of four months with no recurrence, while, on the other hand, Numbers 7, 10 and 13 have lasted six, four and seven years, respectively. In two of these cases there may have been a source of reinfection, for in Case 10, while no history of a discharge in the parents could be obtained, the mother stated that she had a "tube which must be removed," and in Case 7 both parents had a discharge previous to both attacks which the child had. Still it is interesting to note that in the second attack this case acted much more like a chronic one than an acute one. There was only a very slight discharge three weeks after the onset, which is an unusually rapid clearing up of a first attack without treatment, and this discharge, slight as it was, refused to clear up entirely for seven months.

Several children who came to the clinic when it was started two and a half years ago are at present being treated for either a recurrence, or for a discharge which, although very slight, has been continuous. Morrow and Bridgman[19] speak of a child treated sixteen months with 20 per cent. argyrol, fourteen months with 1 per cent. silver nitrate, five months with vaccine, after which the discharge cleared up, to recur again in six months. They also speak of patients treated for two years with the organisms continually present. Epstein[1] thinks it is not impossible for the disease to last eight or nine years.

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18. Schweitzer, Ada E.: Gonorrhea in Children. Indiana Med. Jour., June, 1910, xiii, 257.

19. Morrow and Bridgman: Gonorrhea in Girls; Treatment of 300 Cases. Jour. Am. Med. Assn., May 25, 1912, lviii, 1564.

COMPLICATIONS AND SEQUELAE

The following complications have been observed in seventy-four cases:

	No. Cases
Proctitis	6
Cystitis	5
Arthritis	4
Pelvic peritonitis	1
Inguinal adenitis with suppuration	1
Vulvovaginal abscess	1
Ischio rectal abscess	1

One case had chancroidal ulcerations as well as the vaginal infection. Sheffield,[20] in 148 cases reports the following:

	No. Cases	Per cent.
Proctitis	3	2.0
Arthritis	4	2.7
Local peritonitis	4	2.7
Adenitis	12	8.1
Purulent ophthalmia	7	4.7

Hamilton[8] speaks of ten cases of nocturnal enuresis among his cases, and other writers[21, 22] report cases of urethritis and stomatitis.

Jacobi[23] described a case of abscesses of the neck and hand of gonococcic origin following a gonococcal conjunctivitis in a baby 8 days old.

The proctitis in our cases was not severe and cleared up without treatment, as the vaginal infection began to disappear. In one case the discharge was evident at the anal orifice and after thorough cleansing a positive smear was obtained from the rectum. In this case there was also a marked area of irritation about the anus. Ordinarily in these cases, although the mother speaks of the discharge being present on defecation and intimately mixed with the stools, it is difficult to discover the gonococcus because of the large numbers of organisms found in the rectum.

The extension of the infection into the rectum does not seem to have been very commonly noted. Kaumheimer[24] has collected some cases and has made a thorough study of it. Among ninety-seven cases there were six cases noted, and Flugel,[24] among fifty-six cases of vulvovaginitis, by systematic examination, discovered eleven cases, or 20 per cent. Among

20. Sheffield: Vulvovaginitis in Children with Special Reference to the Gonorrheal Variety and Its Complications. Med. Rec. New York, May 11, 1907, lxxi, 767.

21. Dohrn: Ueber gonorrhöische Erkrankung der Mundschleimhaut bei Neugeborenen. Wien. med. Blätter, 1891, xiv, 3387.

22. Rosinski: Ueber gonorrhöische Erkrankung der Mundhöhle Neugeborener. Deutsch. med. Wchnschr., 1891, xvii, 569.

23. Jacobi: In Discussion of Paper by Cotton. Arch. Pediat., February, 1905, xxii, 106.

24. Kaumheimer: Ueber Rectal gonorrhae im Kindesalter. München. Med. Wchnschr., 1910, lvii, 963.

189 cases of gonococcic infections in children, Pollack[25] had seven cases with rectal complications.

Although with so few symptoms as this complication usually exhibits, it may seem of little importance, still we believe that the gonococcus may remain in the mucous membrane of the rectum for months and even years, being an additional cause of the obstinancy of the disease. The following case may illustrate this:

Y. G., aged 8, Case 10, came to the clinic March 9, 1911, for a discharge which she had had for two years. Her sister had a similar discharge at the same time which had stopped in six months and had not returned. This child, however, had been having daily enemata for pin-worms, which had persisted for some time. The mother says that there had always been mucus in the movements since the vaginitis began. From March 13 to November 1, nearly seven and a half months, there had been no vaginal discharge and no gonococci present in the vaginal secretions, but during this time although there was no further evidence of pin-worms, the discharge was continually present in the rectum. In this case the discharge has always returned when the treatment has been stopped.

We believe cystitis is much more common than is realized, but as the urine is not examined in every case, it is often not discovered. We have had several cases in which there was urethral as well as vaginal discharge from the onset, and in which the urethral glands showed the presence of the infection for a long time after the cystitis and vaginitis had cleared up. This was evidenced by a negative urine and a drop of pus, or later, merely a slight stickiness at the urethral opening. These cases have made good progress on hexamethylenamin (urotropin), but it has had to be continued for some time as the cases relapse if it is discontinued too soon.

The cases of arthritis were of short duration, with one exception. In one case one wrist alone was affected, in another the hip, in another the wrist, elbow and sternoclavicular junction, while the fourth case began in both ankles, then extended to the knees and later to the neck. These cases, especially with regard to the pain, responded well to vaccines.

In the last case, which had become chronic before coming to the clinic, vaccine was given twice a week, beginning at twenty million and increasing ten million each time, according to Hamilton's[8] method. No benefit was noticed until the dose reached one hundred and forty million, from which time it was increased twenty or twenty-five million each time until four hundred million was reached. This patient always received benefit from the vaccines and was decidedly worse whenever treatment was stopped.

The other three cases responded to a few injections of vaccine, the dose being from twenty to fifty million. Plummer[26] reports good results

25. Pollack: The Acquired Venereal Infection in Children. Bull. Johns Hopkins Hosp., May, 1909, xx, 142.

26. Plummer: An Interesting Case of Gonorrheal Arthritis Treated with Serum. Vermont Med. Month., March 15, 1909, xv, 71.

regarding pain in arthritis by vaccines, but found sixteen injections necessary in most cases. In our first case, however, twenty-four injections were given extending over a period of seven months.

Although we have seen no cases of urethritis in male children, many cases have been reported. Cotton[27] reports a hospital epidemic of nineteen cases of vulvovaginitis which probably started from a specific urethritis in a 2-year-old boy. Holt[10] reports a case of gonococcus urethritis in a male child of 2 years; Gordon-Salkind[28] reports two cases of urethritis among fifteen cases of gonococcal infections of children which she has observed, and was able to find only five other cases mentioned in the literature. Wolbarst,[29] in a very comprehensive article, concludes that although it is far less common than vaginitis, it is not a rare condition. He also says that these boys are liable to all of the extensions and complications that occur in the adult and believes that it is possible that many cases of sterility, sexual neurasthenia or urethral stricture are due to a gonococcal infection in childhood. The treatment is practically the same as in the adult.

Holt[10] describes a case of gonococcic arthritis and general pyemia in a boy of 5 years, who was in the same ward with two children with vaginitis and who died of meningitis; also two other cases of meningitis occurring in cases of gonococcal arthritis. These cases, unfortunately, were not proved.

Marfan[5] says that peritonitis is very common and very serious; that it is a general peritonitis, unlike the perimetritis and salpingitis of the adult, with sudden onset accompanied by chills, temperature, vomiting and sharp pains in the abdomen, especially below the umbilicus, and marked cyanosis of the extremities. Although the cases appear to be desperate, he says recovery follows in from eight to twelve days in almost every case, and that it is not necessary to operate.

The case of peritonitis which we have seen was not fatal, but subsided after five days treatment in the hospital, without laparotomy. It may be interesting to know the course of one of these cases. The following history was given on entrance to the hospital:

Seventeen days previously the child waked up with sharp pain throughout the left side of the abdomen. The attack lasted all night and the pain was very severe. The next day she had ten or twelve attacks lasting two or three minutes. For the next thirteen days the pain did not change much in severity but extended throughout the abdomen. For the previous three days the pain was more severe and attacks more frequent. There was no vomiting at any time. The temperature

27. Cotton: An Epidemic of Vulvovaginitis Among Children. Arch. Pediat., February, 1905, xxii, 106.

28. Gordon-Salkind: Ueber Gonorhöea bei Kindern. Inaugural Dissertation. Zurich. Selnau.

29. Wolbarst: Gonorrheal Urethritis in Male Children. Med. Rec. New York. Oct. 29, 1910, lxxviii, 766.

on admission to the hospital was 100.6 F., and gradually dropped until it reached normal on the fifth day. The examination of the abdomen showed tenderness in both iliac regions. No masses were felt but the abdomen was tense and tympanitic. There was a vaginal discharge for which she was treated with long hot douches of boric acid, followed by the instillation of argyrol into the vagina. On the third day of treatment there was considerable discharge after which the temperature became normal. This case had probably passed the acute stage before coming into the hospital.

Epstein[1] reports in detail four cases of gonococcus peritonitis terminating fatally, with either laparatomy or autopsy in each case. In these cases the ages of the children ranged from four to seven years. He mentions three other fatal cases and gives references to many others. Huber[1] is quoted as having seen many cases of subacute peritonitis following a gonorrheal vaginitis. Abt[30] says that tubes, ovaries and peritoneum may be involved and that gonococcal pus was found at autopsy in the tubes of five girls between 7 and 9 years of age.

It is a question still brought forward whether gonococcic vaginitis in children is not merely a local infection without the complications seen in adults. Certainly even a few authentic cases in children in which gonococcic pus has been found in the uterus, tubes, ovaries and peritoneal cavity give the disease a serious aspect. Morrow and Bridgman[19] believe the infection in children may have any of the complications seen in adults. Added to this we have the many resulting cases of arthritis and the possibility of cardiac involvement.

Many authors believe that the disease may later be the cause of malformations of the uterus, dysmenorrhea or even sterility, and that some pus tubes of uncertain origin in virgins may be due to a latent gonococcic infection acquired in childhood. However, we have been unable to find any cases cited in the literature to substantiate this, but it is still a question whether it may not remain latent for years and later cause complications in the developed organs which the undeveloped ones escaped.

TREATMENT

We have tried many different combinations and methods in the treatment, but no specific has been found. The treatment which has given the best results in our cases is local treatment plus vaccine treatment. The local treatment corresponds to the treatment given a case of specific ophthalmia and consists in a cleansing douche followed by the instillation into the vagina of some preparation of silver. The usual directions given the mother are to use a two-quart fountain syringe in which there is a saturated solution of boric acid, and to which the smallest rectal tip is attached. This is to be given three times a day for at least ten minutes,

30. Abt: Gonorrhea in Children. Jour. Am. Med. Assn., Dec. 17, 1898, xxxi, 1474.

as warm as the child can stand it, and with hot water added to it, that the temperature may be kept up. Following this three times a day, either argyrol 25 per cent., or a solution of another silver salt 1 to 1,000, is instilled into the vagina with a small rubber ear syringe. Added to this treatment we have given gonococcic vaccine, beginning with doses of fifty million and increasing twenty-five million, until four hundred million is reached. The vaccine at present is being given once a week, but previously was given twice a week, but with less rapid increase in the dose.

In getting smears a platinum loop has been found the most efficacious, and we have had very satisfactory results from the Weigert modification of the Gram stain, using a 2 per cent. solution of pyronin for a counter stain. This is the stain used at the Boston Board of Health Laboratory.

It seems useless to try to report the results of any treatment on account of the extreme variance of any set of cases selected. Even with the intelligent cooperation of the mothers, and with treatment attempted early in the disease, it is sometimes impossible to carry it out thoroughly on account of extreme nervousness and apprehension in the child. While the simple instillation of the argyrol or some other silver preparation is possible in almost every case, especially if administered with a soft rubber syringe, the douche is sometimes out of the question. I think it would be only fair to report the immediate results of a few of our acute cases in which home treatment was intelligently and thoroughly carried out.

CASES ILLUSTRATING EFFECTS OF TREATMENT

R. G., aged 2½ years, entered clinic Feb. 13, 1911. Thick purulent discharge; smear positive for gonococcus. February 16, discharge better. Feb. 18, no discharge seen then or afterward. (Duration five days.)

O. M., entered clinic May 1, 1911. Purulent discharge from vagina. Smear shows abundant gonococci. After May 15, no discharge was seen for four months, while patient was under our observation. (Duration fourteen days.)

Number 14 (see table), entered clinic May 22, 1911. Profuse vaginal discharge with positive smear. No discharge seen after May 27. (Duration five days.)

Number 15 (see table), entered clinic May 22, 1911. F. G. Discharge from vagina. Smear positive. No discharge seen after May 31. (Duration nine days.)

Number 16 (see table), entered clinic May 22, 1911. M. H. had had discharge for six months Worse for the last month. It had received no treatment. Smear positive. After beginning treatment no discharge was seen for three months while under observation, with exception of one day when treatment was omitted. Negative smears were obtained as soon as discharge stopped in this case.

These are of course our best results, but it shows what is possible under immediate and thorough treatment.

We are adding to this now in every case tonic treatment of some kind, which we believe is very important. In the majority of cases we have given *Eisenzucker* and have noted favorable results.

In some cases in which there has been very little discharge we have used the silver salt alone without the preceding douche, with good

results, on the principle that the solution could reach the tissues better in its concentrated strength. Morrow and Bridgman[19] have recently published an article in which they state that douches alone are useless, and if used with other treatment prevent the latter's action.

Previous to the above treatment, which we have now been using for more than a year, various methods were tried with much less favorable results. Instillations of lactic acid bacilli grown in milk or suspended in bouillon and in sodium chlorid solution were given a long trial. Dextrose was used in different strengths, 5, 8 and 10 per cent. We have used Condy's solution for irrigation in the clinic for a long time, while boric acid was being used at home.

Dr. Lantz and Dr. Cooper, who had charge of the clinic for the first six months, and to whom we are indebted for the use of their records and results, were able to pack the vagina in the older children with argyrol, using a urethral speculum and tape tampons. That it shortened the length of the disease, however, is difficult to say.

Solutions of protargol and argyrol were both used in varying percentages, the argyrol being used as high as 50 per cent. However, no more beneficial results were noted from that than from the 25 per cent. solution.

VACCINE TREATMENT

We have used the gonococcus vaccine in thirty-five cases. Fourteen of these cases have been acute cases, and in this class have been included all cases with profuse discharge of a month's duration or less. Six have been subacute (from two to four months' duration); fifteen have been chronic. In all, there have been 399 injections, making an average of eleven doses for each case, while in reality some cases received as many as thirty-six injections. The average length of treatment for each child was three months, while some treatments extended over eight months. Twenty of these cases still had a discharge, in some cases intermittent, and in many cases very slight, when they were lost sight of. In seven cases there was still a positive smear, although the discharge had cleared up. In only six of the cases did we get both negative smears and an entire clearing up of the discharge, and three of these had a return some time within two years. So that in our hands vaccine has not proved a specific. At first we followed the dosage according to Hamilton.[8] Later on we believed we had better results from larger doses, begun (as mentioned above) with fifty million and increased twenty-five million at each dose until four hundred million was reached. However, we do feel it is an additional factor for good, and believe, with Hamilton and Cooke,[17] that it may shorten the late stages, although, contrary to their results, we have not found the effect so evident in the chronic as in the acute cases. Churchill and Soper[15] conclude that while vaccine treatment

may shorten the stay in the hospital, it does not necessarily shorten the disease. We feel in all cases, however, that it causes much steadier improvement than the local treatment alone, and should be carried out with the local treatment whenever it is possible. Some cases have been treated by vaccine alone, according to Hamilton's recommendation, but the results were not satisfactory and the majority have had it in combination with local treatment of some kind.

In one group of cases we tried some autogenous vaccines from the various other organisms in the vagina. Among these were streptococci, colon, staphylococci and diphtheroids. The results, however, were not remarkable.

We are very much indebted to the laboratory of Tufts Medical School for furnishing us with vaccines, and especially to Dr. Blanchard, who very kindly prepared the autogenous vaccines for us.

IMPORTANCE OF PREVENTION

The following paragraph by Holt has been quoted before by Huber[31] in a discussion of Ward Problems:

> In a disease so difficult to cure and so highly contagious the utmost importance must be attached to measures of prevention. In the light of our experience two things are essential in institution practice; first, cases of gonococcus vaginitis must so far as possible be excluded. In the second place, if admitted by accident or otherwise, they must be quarantined. In excluding cases only one thing can be depended upon, viz., microscopic examination of a smear from the vaginal secretion before the child is received.

The extreme contagiousness of the disease and the great danger of infection in hospitals is a fact much better known to hospital superintendents and superintendents of nurses than it is to physicians in general. Holt's[10] account of the experiences at the Babies' Hospital in New York makes one realize how difficult the disease is to control even under the most careful preventive measures possible. However, they have at last been able to control it, but only by the exclusion of all suspicious cases.

Perhaps the three most important items of the routine which has been carried out in the Babies' Hospital in New York for several years are as follows:

> 1. Vaginal smears at entrance and once during the week throughout the hospital.
> 2. Individual thermometers, and individual bottles of liquid petrolatum for a lubricant.
> 3. The disinfecting of nurses' hands in going from case to case.

At the Children's Hospital in Boston the extra precaution is taken of bathing the genitalia of each child with a weak solution of bichlorid before the bath is begun. It is also a rule of the hospital that every towel

31. Huber: Ward Problems. Arch. Pediat., September, 1911, xxviii, No. 9.

must be sent to the laundry after having been used once. Here also the nurse disinfects her hands before going to each new patient. Since instituting these precautions they have been entirely free from epidemics of this kind.

A school nurse recently asked if all the cases should not be excluded from school. Although this may be done during the acute stage, it seems impossible to prevent children from going to school for many months, or perhaps years, from this cause. After the acute stage we have allowed our children to go to school, provided they did not use the toilet. Where there is a school nurse it would seem possible to have special precautions taken with these cases.

In Chicago this condition has already been provided for in the Frances Juvenile Home. There, under the direction of Dr. Clara P. Seippel, children with venereal disease are treated, and, at the same time, continue their education. A new building is about to be erected in which several hundred children may be cared for in this way. Dr. Seippel[14] says:

> The Frances Juvenile Home was founded in 1909 for the purpose of treating and educating while under treatment girls who have passed the acute stage of gonorrheal vulvovaginitis and children who have hereditary syphilis. A school room and a teacher are provided by the Board of Education, and older children receive practical instruction in domestic science.

Besides this home, Chicago is fortunate in having a children's venereal ward in the Cook County Hospital, where medical and surgical disorders plus a gonococcal infection may be treated. This, it seems to us, should be instituted in every large city.

Dr. Meigs and Dr. Seippel found that during the year 1910 over five hundred cases of gonorrheal vulvovaginitis in children had passed through twelve different hospitals and dispensaries in Chicago.

Dr. Pollack[25] estimated that there were from eight hundred to one thousand cases in Baltimore each year. This estimate, however, was based on only about 130 actual cases. However, the disease is much more prevalent than is generally realized.

That one of the parents may be the cause of this disease oftener than we realize suggests the necessity of emphatic prophylactic warnings in all cases of gonorrhea in adults of either sex, in the latent as well as in the active stage. Schweitzer[18] believes that latent gonorrhea in either parent may become a source of infection to the other and later to the child.

CONCLUSIONS

1. We believe that all cases of vaginitis with a persistent discharge, which at any time has been profuse, are due primarily to the gonococcus.

2. That the disease may extend over many years, during which time there may be many recurrences, and that the periods of latency may at least be as long as eighteen months.

3. That vulvovaginitis in children, although it may remain a local disease, is liable to any of the complications seen in adults.

4. That the most efficient treatment does not insure a permanent cure.

5. And finally, that physicians should realize the importance and prevalence of the disease and institute stricter preventive measures, both in hospitals and in private practice.

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II. THE PANCREATIC FERMENTS IN INFANTS IN ACUTE INTESTINAL INDIGESTION (ALIMENTARY INTOXICATION)*

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In a paper recently published in this JOURNAL,[1] we reported on the pancreatic ferments of infants. For this study the duodenal catheter was employed and pancreatic juice was obtained for examination directly from the duodenum. It was therefore not necessary to have recourse to the previous indirect methods, such as tests of stools or extracts of the pancreas. A large number of cases were examined by this method; the report including normal infants of various ages, as well as sick infants suffering from pylorospasm and from malnutrition. It seems unnecessary to review the results obtained. For the present purpose, it is sufficient to repeat, that the three pancreatic ferments investigated, namely, trypsin, amylase and lipase, were found almost constantly present even in advanced cases of marasmus (atrophy, decomposition).

The large series of cases of the first report did not include any examination made on infants suffering from *acute* disturbances. Although numerous tests of this nature had been carried out, they were deemed at the time insufficient in number to be classed as a group, and were therefore not brought forward for consideration. Since then, however, other cases of this description have been tested, almost all suffering from acute intestinal indigestion of moderate or severe grade. From the first beginning of catheterization of the duodenum, which now dates back over two years, two groups of sick infants seemed to offer the greatest promise for experimental investigation of the pancreatic ferments, that of malnutrition which has been already considered, and another disease which is likewise characteristic of infancy and one of the largest factors in infant mortality, variously termed acute intestinal indigestion or alimentary intoxication.

In this further paper on this subject we report on examinations of cases of the second group, acute intestinal disturbances. The number of cases which form the basis of the present study is not large, but as the seasonal incidence of the disease would necessitate our again waiting for

*From the Research Laboratory, Dept. of Health, New York City. Presented before the Society for Biology and Experimental Medicine, February, 1912.

1. Hess, A. F.: AM. JOUR. DIS. CHILD., October, 1912.

a long period before more cases presented themselves, it seemed advisable to publish some interesting data on this important topic.

The technic employed was in all respects the same as in the previous investigation of pancreatic ferments. For the trypsin test, gelatin and egg albumin were used, for amylase a 1 per cent. starch solution, and for lipase neutral ethyl butyrate. We realize that these methods do not furnish quantitative tests for these three ferments, but afford merely an index of ferment activity. It is realized, likewise, that all quantitative estimations of ferments are fraught with error. In view of this inherent deficiency of our present methods, the subject was regarded in the previous study almost entirely from a qualitative point of view. However, the estimations to be reported, when considered collectively, vary so widely from those obtained on normal infants, or indeed from those suffering from chronic malnutrition, that a comparison on the basis of quantitative results seems justified.

Thirteen acute febrile cases were tested by means of the catheter, comprising a total of eighteen tests. These infants varied in age from 6 weeks to 9 months. Of the thirteen, all but three were cases of acute intestinal indigestion (alimentary intoxication). The three non-intestinal cases included pneumonia, empyema and adenitis, and are considered with the others to advance the question whether the results which we obtained are attributable to the acute intestinal disturbance or are common to all febrile conditions.

For purposes of comparison, we have framed our results in a table (Table 1), which affords a clearer picture than mere description. The tabulated cases have been divided into four groups: (a) cases of acute intestinal indigestion, (b) other febrile cases, and two groups of chronic disturbances, (c) marasmus, and (d) marasmus accompanied by pancreatic succorrhea. In all groups, it is seen that tryptic activity was adequate; this ferment has always been found the most constant. When we review our results of amylolytic activity, although we find noticeable differences in the figures obtained, they are not sufficiently marked or constant to draw distinctions.[2] It is different, however, in regard to lipase. In almost all cases of acute intestinal disturbances the lipase was found to be deficient, in some cases almost absent. It should not be thought that this was merely a premortal phenomenon, for, although most of the infants were suffering from severe disturbances, all but three

2. It should be remembered in this connection that the higher figures denote a smaller and not a larger amount of amylase, as they refer to the volume of starch solution (acted on for forty-eight hours by the amylase), which it was necessary to add in order to completely reduce the Fehling solution. The reverse is true as regards the figures for lipase; they bear a direct relation to the amount of fatty acid formed during the incubation of the neutral ethyl butyrate with the ferment. The details of these tests are given in the previous paper.

recovered. The cases of marasmus which we record (c) were in a far more critical condition than these acute cases. Their respective ages and weights are appended to show the extreme degree of their atrophic condition. Nevertheless, all the pancreatic ferments, including lipase,

TABLE 1.—TESTS OF PANCREATIC FERMENTS

Cases	Age, Mos.	Lipase	Amylase	Trypsin	Remarks
A. CASES OF ACUTE INTESTINAL INDIGESTION (ALIMENTARY INTOXICATION)					
1	2	1.7	30.6	24 Hours	.....
2a	..	0.9	..	.....	.....
b	3	0.7	50	24 Hours	Death 48 hours after second test.
3	2½	3.2	46.5	24 Hours	Mild Case.
4	3½	1.8	50	48 Hours	.....
5a	...	1.3	42.0	24 Hours	.....
b	6	1.0	48.5	.....	.....
6	1½	0.3	50	24 Hours	Death after 24 hours.
7a	...	1.4	50	24 Hours	.....
b	4	0.1	..	24 Hours	.....
c	...	1.6	41	.....	.....
8	9	1.4	50	48 Hours	.....
9a	5	1.2	39.5	24 Hours	.....
b	...	1.6	48	.....	.....
10	4	0.4	50	24 Hours	Death after 48 hours.
B. OTHER FEBRILE CASES					
11	4	3.3	....	.....	Pneumonia.
12a	...	2.4	39.5	.....	.....
b	6	4.2	48.0	.....	Empyema.
13	4½	5.1	43.5	24 Hours	Cervical adenitis.
C. MARASMUS					
14	6	3.0	31.0	24 Hours	Weight, 6 pounds, 15 ounces.
15	16	8.7	17.3	Digested Egg White	Weight, 9 pounds, 3 ounces.
16	4	3.8	30.3	Digested Egg White	Weight, 6 pounds, 8 ounces.
17	2	4.8	25.0	.....	Weight, 6 pounds, 8 ounces.
18	2½	3.4	50	24 Hours	Weight, 6 pounds, 2 ounces.
D. MARASMUS ACCOMPANIED BY PANCREATIC SUCCORRHEA					
19	2½	0	50	24 Hours	A second test also showed absence of lipase.
20	2	7.8	50	24 Hours	20 c.c. of golden alkaline fluid aspirated in 50 minutes.
21	5½	0.9	33.5	48 Hours	45 c.c. of yellow alkaline fluid aspirated in 75 minutes.
	...	0.9	29.3	24 Hours	

were generally present in normal amount. In the acute intestinal cases the deficiency of lipase seemed to bear to some degree a direct relation to the severity of the illness. In the mildest case which we tested, the lipolytic action was merely somewhat reduced. Furthermore, in a case showing the classical symptoms of alimentary intoxication (No. 7),

including the stupor and glycosuria, in which we carried out three tests, we obtained far larger amounts in the early stage of the illness and during the period of beginning recovery than in the test made during the height of the illness.

It seems unnecessary to give in detail the clinical history of the various cases; however, in order to give an idea of the type of case referred to, we shall summarize the clinical history of a few:

CASE 1.—M. C., aged 2 months, entered the hospital with a history of five days illness accompanied by fever, slight vomiting and marked diarrhea; 6 to 10 watery stools a day. Child was moderately nourished; weight 9 pounds, 8 ounces. Prostrated. Typical appearance of cholera infantum. Treatment *Eiweissmilch*. Recovery.

CASE 2.—Aged 3 months. Very similar to the previous case. Death forty-eight hours subsequent to examination.

CASE 3.—Aged 2½ months, suffering for about ten days from enteritis; six to seven stools a day, containing undigested food.

CASE 4.—Aged 3½ months; weight 9 pounds, 4 ounces. Well until forty-eight hours previously. Acute onset with vomiting and diarrhea. Temperature between 103 and 104 F. Moderate diarrhea; no vomiting in hospital.

The question naturally suggests itself as to whether the deficiency of ferment secretion may not be dependent merely on the febrile condition. With this doubt in mind some non-intestinal febrile cases were examined. In a test made on an infant suffering from pneumonia and recording a temperature of 104.3 F., in another admitted to the hospital for empyema with a temperature of about 103 F., and in a third which had cervical adenitis and a temperature ranging for some days between 101 and 104 F., it will be seen (Table 1 B) that lipase was not found deficient. It would seem as if there were something distinctive in the relation of the lipase deficiency and the intestinal disturbance. There is a group of cases which was described in the former paper under the designation of "pancreatic succorrhea," in which a low lipolytic activity was frequently found (Table 1 D). In these cases, which have been encountered among atrophic infants, there is a marked hypersecretion of watery pancreatic juice, which is especially weak in lipase. With the exception of this group in which the lack of lipolytic activity must in a large measure be offset by the increased quantity of pancreatic fluid secreted, we have not met with instances manifesting this deficiency.

DISCUSSION

It would be unwise to draw broad deductions from these tests, as further examination may show that in some instances lipase is not lacking. However, in view of the fact that this ferment alone of the three pancreatic enzymes, was found lacking in acute intestinal disturbances, it may be in place to add a word as to some closely related clinical and metabolic manifestations of this disorder. Clinicians are agreed that

infants suffering from acute intestinal derangements tolerate fat poorly, and indeed only slowly regain the power to assimilate this food-stuff. We do not wish to insist too closely on this parallelism of lack of lipase and lack of toleration for fat; however, the experimental fact fits in so closely with clinical experience that one cannot refrain from suggesting the correlation. It may well be that this fat intolerance is based on more complex functional disturbances. With this same qualification it should be remembered that a study of the metabolism of alimentary intoxication[3] has shown that in this condition, even after the acute period, there is poor absorption of fat and an increase of neutral fat in the stools.

CONCLUSIONS

In the former communication, which considered the pancreatic secretions in chronic malnutrition in infants, it was found that the various ferments of the pancreas are normally secreted even in advanced instances of marasmus or atrophy. In the present study of an *acute* disease, of acute intestinal indigestion, or alimentary intoxication, which was carried out also by the direct method, by the use of the duodenal catheter, the lipase was found deficient, although the two other pancreatic ferments were present in considerable amount. The deficiency of lipase seemed to be to some degree characteristic of this disturbance; it is not a general characteristic of all febrile conditions, and was not met with in pneumonia or empyema. It is possible that the lack of lipolytic activity in this disease should be correlated with the clinical manifestation of fat intolerance, and the metabolic studies showing a deficient absorption of fat.

3. Meyer, Ludwig F.: Jahrb. f. Kinderh., 1907, lxv, 585.

PROGRESS IN PEDIATRICS

RECENT CONTRIBUTIONS TO THE STUDY OF PYELITIS IN INFANCY

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The term pyelitis is used to designate an acute febrile disease characterized by the presence of pus in the urine. Brennemann[1] estimates that these cases constitute 1 per cent. of all those coming to a physician. The symptoms are usually abrupt in onset, the temperature often reaches 103 or 104 F. The fever may be relapsing in type[2] extending over variable periods of time, sometimes simulating malaria.[3] Rigor may be present and is almost pathognomonic of this disease when it occurs in infants under 2 years of age.[1, 4] The nervous symptoms may suggest meningitis.[4] Sometimes there are definite symptoms pointing to the genito-urinary tract, as frequent micturition accompanied by more or less pain,[4, 5] but more frequently there is nothing to indicate any disturbance in these parts.[1] The physical examination is usually negative except that pallor is characteristically present.[1] In some instances the kidney is palpable,[1] more often the right than the left, and often the spleen is palpable at the onset of the disease.[4, 6] There may be tenderness in the lower abdomen[4] or back, which makes the patient object to being handled.[1] Occasionally there is pain and tenderness of the joints with signs of local inflammation.[4] Herpes may be present.[4] Occasionally there is an inflammatory condition of the vulva.[4]

Urine: A single specimen of urine may be normal. The sediment may contain only bacteria,[7] but usually shows also many leukocytes. The urine may be opalescent, due to the presence of microorganisms.[4, 8] The reaction is acid when fresh, but becomes alkaline on standing.[9] The amount of albumin is slight.[6] The presence of casts indicates that the

1. Brennemann: Jour. Amer. Med. Assn., 1911, lvi, 631.
2. Wassermann: Am. Jour. Med. Sc., 1911, cxliii, 878.
3. Vanderhoof: Jour. Am. Med. Assn., 1912, lviii, 1172.
4. Box: Brit. Med. Jour., 1910, ii, 1128.
5. Rawls: Med. Rec., New York., 1911, lxxx, 709; Med. Rec., New York, 1912, lxxxi, 359.
6. Dudgeon: Lancet, London, 1908, i, 615.
7. Porter and Fleischner: Arch. Pediat., 1910, xxvi, 826.
8. Thompson, W. H.: Med. Rec., New York, 1910, lvii, 907.
9. Thompson, T.: Quart. Jour. Med., 1909-10, iii, 251.

parenchyma of the kidney is involved, but their absence does not necessarily mean that the kidney is normal.[4, 6] Pus may be temporarily absent from the urine when only one kidney is diseased, due to a blocking of the ureter.[1, 9] The organisms responsible for the infection are colon bacilli in a very large percentage of the cases.[9] Jeffries[10] reports 121 cases, 67 colon, 37 staphylococci, 10 streptococci, 3 pneumococci and 4 miscellaneous. The colon bacilli when present are extracellular and show a tendency to beading.[1, 6]

PROGNOSIS

The course of the disease is very variable. Usually the acute symptoms are over in a few weeks. A considerable number of cases tend to become chronic and may last for years with acute exacerbations. Birk[11] reports one child who had five recurrences in ten years, the last at the age of 12. Not infrequently the acute attacks come in storms with periods of comparative health between.[12] Some infants have attacks like periodic vomiting and may pass into coma.[4] The ultimate outlook in nearly all cases is favorable, but the rapidity of the cure depends largely on one's definition of "cured." The more favorable statistics come from those who do not require that the urine be culturally free from bacteria before calling the patient cured. Thompson[8] believes that chronic interstitial nephritis may result because of the slow infection of the kidney by colon bacilli.

PATHOLOGY

Pathologically two types of the disease may be distinguished.[1] The first shows simply a catarrhal condition of the bladder and the pelvis of the kidney. The second, which occurs especially in children with low resistance, shows multiple abscesses in the cortex of the kidney.[13] Most of the autopsies are in this type. Sometimes these abscesses radiate toward the pelvis in fine lines. The bursting of these abscesses gives rise to pyuria.[8] Müller[14] believes that the infection from the pelvis of the kidney in these cases travels by way of the lymphatics and that the tubercles play a secondary rôle. Very few autopsies have been made, because the condition is so rarely fatal. Apparently no permanent damage to the kidney results in cases which are cured. Friedenwald[15] reports one case in which the patient died of tuberculosis seven weeks after a cure of pyelitis and there were no lesions in the genito-urinary tract.

10. Jeffreys, W. M.: Quart. Jour. Med., 1911, iv, 267.
11. Birk: München. med. Wchnschr., 1912, lix, 1429.
12. Hutchinson: Clin. Jour., 1911, xxxviii, 209.
13. Thiemich: Jahrb. f. Kinderh., 1910, lxxii, 243.
14. Müller, A.: Ztschr., f. Urol., 1912, Suppl.

DIAGNOSIS

The diagnosis of pyelitis can be made only by an examination of the urine. Agglutination reactions and opsonic index determinations are of no value.[6]

CHANNEL OF INFECTION

The mode of infection has been the subject of much controversy. Birk[11] believes that a coexisting tuberculosis and the so-called exudative diathesis are predisposing factors. Brennemann[1] has called attention to the fact that it occurs more frequently in the summer months. Friedenwald[15] showed that fifty-nine out of eighty cases had preceding gastro-intestinal disturbances. Very frequently cases are preceded by acute nasopharyngeal infections.

The fact that such a very large percentage of cases occur in girls has given support to the theory of an ascending infection. The disease is most common in the diaper age, when there is great possibility of contamination of the urethra with fecal matter. Against this mode of infection has been cited the fact that rarely is there any cystitis present in these cases and that in vulvovaginitis of infants cystitis is rare.[16] Leutscher[17] has shown that bacilli may be present in the bladder without a cystitis, so that an infection of the kidney might occur. Dudgeon[6] has also reported twenty cases of constipation, two of which had colon bacilli and pus in the urine, but no cystitis or other constitutional symptoms. The disease occurs in boys following circumcision, which is taken to indicate an ascending infection.[18] It is possible, as Thompson[9] has pointed out, that the infection is ascending, but the lumen of the ureter is less important than the deep tissues around it and around the urethra. Stewart[19] has shown experimentally that the vessels of the ureter usually carry infection, not the ureter itself.

Another theory of the mode of infection is that the bacteria are borne to the kidney by the blood-stream. Presumably the bacteria enter the blood-stream from the intestines or possibly through some break in continuity of tissue about the rectum. In order that these bacteria shall do harm there must be a lowered resistance of the kidney or an injury to the tissues of the kidney, but this injury may be caused by the toxins liberated from the bacteria in the blood.[13] Apparently certain healthy persons take bacilli coli from the intestines and excrete them through the kidneys.[5] The number and virulence of these organisms depends on the

15. Friedenwald: Arch. Pediat., 1910, xxvii, 801.
16. Walker, J. W. T.: Practitioner, 1911, lxxxvi, 655.
17. Leutscher: Bull. Johns Hopkins Hosp., 1911, xxii, 361.
18. Rolleston: Practitioner, 1910, lxxxiv, 439.
19. Stewart: Univ. of Penn. Med. Bull., 1910, xxiii, 233.
20. Collica: New York Med. Jour., 1911, xciv, 376.

intestinal condition. Anything which interferes with free excretion of the urine tends to produce an infection of the kidney. Experimentally it is possible in certain instances for the bladder to become infected by this means and the kidney itself remain normal.[21]

A third possible mode of infection is the direct transmission of bacteria from the intestines to the kidney. Probably this does not occur unless there is some damage to the lumen of the intestine.[13] It has been shown[21] in rabbits that the ascending colon and cecum are connected with the right kidney by a chain of lymphatics. None are found connecting with the left kidney. If only one kidney is affected by infection it is more frequently the right.[10] With even a slight damage to the intestinal wall, bacteria can pass by the lymphatics directly into the kidney. There is also a free anastomosis between the lymphatics on the side of the vertebral column and those of the ureter so that an infection might easily occur from some outside source.[22]

The consensus of opinion seems to be in favor of the ascending method of infection in the majority of the infants under 2 years of age. The condition in these cases is rarely anything more than a catarrhal pyelitis. In children beyond 2 years of age the other means of infection come into play and not infrequently the severer types of lesions with multiple abscesses are found.

TREATMENT

Spontaneous cure occurs in many acute cases.[23] Many of the chronic cases are resistant to all means of treatment. Box[4] says the value of any treatment is to be measured by its action in chronic cases. In the acute cases there seems to be the greatest uniformity in the belief that the urine should be made alkaline and kept so for a considerable period of time, and that free diuresis should be established.[24] Colon bacilli are believed to develop less easily in an alkaline urine and phagocytosis of the bacilli by the leukocytes is more active in an alkaline medium.[25] Potassium citrate or sodium bicarbonate are the drugs most used in this connection. Directly opposed to this view is the observation of Betz,[26] that the thin, weakly acid urine occurring normally in pyelitis is the most favorable culture medium for the colon bacillus, but that it is unable to proliferate in very acid concentrated urine. He gives phosphoric acid and a meat and no-vegetable diet in order to make the urine strongly

21. Franke: Mitteil. a. d. grenzgeb. d. Med. u. Chir., 1911, xxii, 623 and 1911, xlviii, 1973.
22. Clarke, W. B.: Clin. Jour., June 28 and July 5, 1911.
23. Chambard: Ann. d. méd. des org. G. U. 1911, xxix, 865.
24. Pardoe: Brit. Med. Jour., 1910, ii, 1129.
25. Dick: Brit. Med. Jour., 1910, ii, 1134.
26. Betz, Meyer: Deutsch. Arch. f. klin. Med., 1912, cv, Nov. 5-6.

acid, and limits the intake of fluids and gives hot air baths to produce concentration.

The statements in regard to urinary antiseptics would make one feel that on the whole they are not of great value in the acute cases and worthless in the chronic cases. Ammonium and sodium benzoates,[24] calomel,[12] sandalwood oil,[24] creosote,[27] salol[15] and urotropin are among the drugs recommended. Hexamethylenamin, combined with a sodium salt is said to be more efficacious than the simple hexamethylenamin.[28] The colon bacillus easily becomes accustomed to any drug given internally, so a change of drugs should be made frequently.[26] Nevertheless, hexamethylenamin is the favorite antiseptic drug. In this connection it is important to bear in mind that the action of hexamethylenamin depends on its being broken up in the body and excreted as formaldehyd. Sisson[29] and others have shown that the excretion of formaldehyd takes place only in an acid urine. It has been customary to give hexamethylenamin in connection with alkalies, and this may account for the unsatisfactory results from its use. Thompson recommends that it should be given always with an equal amount of sodium benzoate.[8] Box[4] has shown that hexamethylenamin has very little influence on the colon bacillus, and Dudgeon[6] goes so far as to say that if a patient with pyelitis is benefited by hexamethylenamin, the offending organism is not a colon organism.

Vaccines in the acute and subacute cases do not appear to influence the result obtained by other methods of treatment.[4, 12, 24, 30] They are of no value in chronic cases. Vaccines do not prevent exacerbations, nor affect the presence of pus and blood in the urine.[31] In some cases they apparently give relief of symptoms.[32, 33, 34, 35] Lyman says[36] that autogenous vaccines are of great value, but adds, "many cases do not improve more rapidly than others without it." Rolleston reports favorably on their use.[18] Dick[25] believes that cases which do not respond to alkaline diuresis are sometimes benefited by vaccines. Serum apparently has no bactericidal action on the colon bacillus. Normal human serum and immune horse-serum are identical in this respect.[6]

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27. Briscoe: Lancet, London, 1909, ii, 1269.
28. Casper and Citron: Ztschr. f. Urol., 1911, v, 241.
29. Sisson: Unpublished communication.
30. Geraghty: Tr. Congress Am. Phys. and Surg., 1910, viii, 271.
31. O'Neil, R. F.: Am. Jour. Urol., 1911, vii, 209.
32. Williams, R. S., Murray, H. L., Wallace, A. J.: Jour. Obst. and Gynecol. Brit. Empire, 1912, xxii, 65.
33. Williams, Craigin and Newall: Tr. Congress Am. Phys. and Surg., 1910, viii, 148.
34. Cabot, H.: Am. Jour. Urol., 1911, vii, 131.
35. Pringle, S.: Practitioner, 1911, lxxxvii, 35.
36. Lyman, C. B.: Northwest Med., 1911, iii, 31.

Pardoe[24] suggests that in the chronic cases there is possibly some underlying condition, as a disturbance of the gastro-intestinal tract, which must be relieved before the pyelitis will subside. Free evacuation of the bowels is advocated,[27] and the giving of calomel and cold enemas.[12] Some longstanding chronic cases of bacilluria are entirely cured by appendectomy, even though there are no symptoms referable to that organ.[8, 35] Operation is advised in cases in which there is any suggestion of abdominal disease or in those very stubborn to treatment.[10]

No local treatment is indicated in acute cases. In resistant chronic cases bladder washings with various antiseptic drugs may be tried.[24] Koll[37] believes that the best results are obtained by the use of aluminum acetate.

329 Beacon Street.

37. Koll: Amer. Jour. Urol., 1912, viii, 287.

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